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THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 74 DECEMBER, 1955 No. 6

RADIOLOGY IN THE SEVENTH DECADE*

By PAUL C. HODGES, M.D.
CHICAGO, ILLINOIS

I LACK the eloquence to tell you how greatly I appreciate the honor which Dr. Dickinson on your behalf has just bestowed upon me.

As President-Elect I have spent much of my spare time this past year working with the other members of the Program Committee on the Scientific Program, which will start a few minutes hence, and now as President I am allotted a little time in which to speak a few words of wisdom. Those words have been put together and it remains to be seen whether you will consider them wise, but before reading them I want to express the thanks of the entire Program Committee for help we have received from many people. In the time available I cannot name them all but I must mention at least the members who have arranged the symposia, the authors and the discussants of the papers on the Scientific Program, and the director and faculty for the thirty-eight Instruction Courses. Then there are the members and invited guests who have been willing to assume the heavy responsibility of preparing, installing and presenting the scientific exhibits, and the commercial exhibitors whose displays add so much to the importance of the meeting and whose payments for exhibit space defray so much of the expense.

Particular thanks are due to our overseas guest, Professor Wegelius, to the Caldwell Lecturer, Professor Lester Dragstedt, and to Mr. Lawrence Kimpton, Chancellor of The University of Chicago, who somehow has carved time out of an enormously busy schedule in order to be with us today.

It will soon be sixty years since a German physicist made a discovery that brought him the world's first Nobel Prize in Physics and paved the way for the establishment of the branch of medicine in which we who meet here today earn livings for ourselves and families.

The Nobel Prize is a great and fitting recognition of outstanding achievement and it is a praiseworthy attribute that most American radiologists are economically self-sufficient, contributing taxes rather than consuming them; but Roentgen was working without thought of honor or reward, and physicians who are worthy of the name are attracted to radiology for reasons quite apart from the opportunity for financial gain. Roentgen was spurred to his work by a desire to understand the passage of electricity through gases, a phenomenon easily explained to the schoolboys

* President's Address delivered before the Fifty-sixth Annual Meeting, American Roentgen Ray Society, Chicago, Illinois, September 20–23, 1955.

of 1955 but difficult of comprehension even by the most mature minds of 1895; and physicians increasingly are attracted to our field of medicine because even in its seventh decade radiology will continue to present the challenge of innumerable unsolved physical, chemical and biological problems.

Our program has been designed to illustrate some recent successes at solving such technical and biological problems and to point to others still requiring solution, and in the few minutes available to me I would prefer to talk of such matters but I have decided instead to devote the time to consideration of an entirely different sort of problem that has concerned us in the past and will continue to confront us in the seventh decade on which we are about to enter.

Ten years ago, writing of the technical progress that had occurred in the first fifty years of radiology and attempting a few guesses at the developments that would come in the sixth decade,[1] I recognized that there were social problems as well and said that the coming decade was bound to see a change in economy that would affect every phase of our national life. I am sure you will agree that that change did come.

It is certainly true and perhaps fortunate that sociologically speaking physicians tend to be reactionaries, preferring the status quo and resisting social change; but it was inevitable that the economic upheaval following World War II must involve medicine as it has involved every other phase of life in our country, and stability has not yet been reached. If we physicians are to adapt successfully to this inevitable and continuing change, we must understand certain important differences between the social problems of the past decade and those of the decade that lies ahead. Ten years ago politically powerful forces were using medical care as a pawn in a vast game in which the real issues purposely were obscured. The pros and cons of hospital insurance, government support for hospitals, and the increased complexity and cost of medical care were slanted to serve an over-all political purpose, and in the free-for-all fight that followed it was difficult to differentiate friend from foe. Under such circumstances a decision to not move at all at least had the advantage of avoiding movement in a direction that might prove to be the wrong one.

Ten years later, though the irresistible pressure for change continues, the sinister forces that were exploiting it to the disadvantage of medicine are largely dissipated. Radiologists, like other physicians, have devoted money and effort thwarting that exploitation, but the task would have been hopeless without the general setback that has been given to the scheme for widespread socialization of our nation's economy. That job was done by the public at large, who by virtue of their great numbers wield political power of a sort that physicians always must lack. This public, who are our patients, have used their political power not as a favor to us but in enlightened self interest. Increasingly they have come to appreciate that the pursuit of happiness, which is their constitutional right, depends in great part on good health and longevity and that well-trained, dedicated, unfettered physicians are essential to both. Like all of us, they dislike heavy taxes and the rising costs of everything, including medical care, and they expect and have a right to expect that we will do everything in our power to effect economies in our work and pass the savings on to them. They trust us to assist private and government insurance agencies to increase the benefits for hospital and medical care but they no longer believe the myth that by some political legerdemain the finest of medical care can be rendered less expensive than the cheapest kind of second-hand automobiles. As long as we continue to merit their confidence and respect, our patients will provide the political power to protect medicine from those who would use it as a tool, but how do we do this? By being increasingly effective in helping them to avoid or recover from disease and to live in reasonable comfort to ages that were unthinkable a generation ago; by doing all in

our power to keep down the costs of all sorts connected with medical care; by deemphasizing our interest in the financial rewards of our profession and by avoiding acrimonious disputes between physicians themselves and between physicians and the numerous other workers who are essential to the teamwork of modern medicine.

In the days before trench mortars, tanks, and motorized guns, soldiers were taught that infantry advancing against retreating infantry and meeting here and there islands of resistance should endeavor to avoid employing its guns lest the enemy retaliate with guns of its own and an infantry skirmish be turned into a major engagement. The same principle applies in family squabbles and in disputes between neighbors. If the difficulty can be localized, usually tranquillity can be restored; but if tempers are sufficiently roused, minor faults are magnified, bitter things are said and serious trouble follows. At the moment the radiologists and pathologists of a neighboring state supported by colleagues from every branch of medicine are pitted against certain hospitals of that state in a controversy over issues vital to both sides, difficult to compromise and, therefore, deserving of patience and good will on the part of all concerned. Instead, tempers have been allowed to flare, the case has been taken to the courts, and regardless of the outcome public respect for doctors and hospitals is bound to be diminished.

A physician testifying as an expert witness in the pay of the hospitals has made statements about radiology which presumably sprang from anger and the pressure of examination and cross examination and probably would not be believed even by himself under normal circumstances. It is possible to interpret his testimony as indicating that he, all hospital administrators and most physicians consider radiology to be not in fact the practice of medicine but rather a hospital service that should be under the control of the hospital administrator. Of course, the facts are as different as possible. In the past twenty years radiology has attained an unshakable position as one of the most essential phases of clinical medicine. Universities testify to the fact by continuing to create departments of radiology with larger and larger staffs and more and better technical facilities, and our clinical colleagues come to us as they do to one another seeking our help in all sorts of clinical problems. Orthopedists, internists, gastroenterologists, etc., who are worth their salt, have a great deal of skill in the radiology of their particular fields but the abler they are the more regularly they value our assistance. It is the neophyte who contains all knowledge under one scalp and his brief period of self-satisfaction excludes all outside help, not merely ours.

The ablest and most influential among the hospital administrators, those who head hospital associations and conduct university courses in hospital administration see eye to eye with us in this matter. Men of this sort accept as axiomatic the fact that the quality of the radiology that will be practiced in a hospital depends entirely on the quality of its radiologists. If these radiologists are capable, of course they will have demanded and will have received good equipment, an adequate nonprofessional staff, and a free hand in running the show; and, on the contrary, if they are incompetent the finest technical facilities will not compensate for the fault.

If I am correct and if basically there is no serious disagreement between the aims of the hospitals and those of the radiologists, how has the quarrel come about? In part it is because not all hospital administrators and board directors are intelligent and trustworthy and some undoubtedly would like nothing better than to take over the operation of the radiologic departments of their hospitals, with only token aid from the radiologist and with emphasis on net income to the hospital regardless of the effect on the professional quality of the work. But shoddiness is encountered in other walks of life as well and when it is detected and exposed to public view, usually it is easily corrected.

Unfortunately, we ourselves must take

part of the blame for the quarrel that has now assumed such alarming dimensions. To my mind, though our hearts may have been pure our logic has been bad. Some of us, having looked at the office practice of radiology and finding it good, have said that it should be the pattern of radiology in hospitals and medical schools as well. We have observed that a private radiologist renting space in an office building is at once a physician and a businessman and that in the latter capacity he is responsible only to his creditors, his bankers and the tax collector. We find that his patients come to him primarily because they or the family physicians or specialists who have referred them have confidence in his ability and that, though it is true that he enjoys some automatic advantage if many other physicians have offices in the same building with him, still his patients are ambulatory and he will lose them to other radiologists in nearby or even distant office buildings if the latter have more to offer professionally than he. He bills his patients directly and if he is fortunate meets his rent, supply bills and payroll and has something left for his family, and we find this excellent and in keeping with our preference for a free unfettered economy. All this is well and good but then, by means of a few false premises and a few false inversions of the sort so dear to the human mind and so exasperating to the teacher of freshman logic, some of us have arrived at the conclusion that it will be difficult if not impossible for the hospital or medical school radiologist to maintain the proper professional relationship with his patients and exercise adequate control over the acquisition and functioning of his equipment and technicians unless he operates his hospital or medical school department essentially as though it were a private practice in a professional office building. It is conceded that in city, county, state or federal institutions or in the case of free patients in private hospitals, the patient-doctor relationship may be professionally correct and the control of the department adequate even though the radiologist is salaried, but for some reason that is not easily explained it is argued that salaries become unethical when the patient is able to pay a fee.

Actually, most radiologists do not reason this way. We find it easy to distinguish ethics from economics and to distinguish between certain radiologist-hospital arrangements that are in fact ethical and others that are not. We believe that variation in economic arrangement is inevitable and desirable in a free society and consider it only natural that salaries should be the arrangement in many universities and clinics, a percentage arrangement the most desirable plan in many non-teaching hospitals, including those operated by certain church groups, and a rental basis the one preferred in some other institutions. Unfortunately, however, our record of reasonableness is unknown to many of those who hear of our occasional unreasonable insistence on the sacredness of the method of rendering the fee and we appear to such persons to be insisting that radiologic ethics have to do largely if not entirely with money. Somehow, means must be found to correct this mistaken opinion and to make known the real issues. These issues are two in number:

(1) Hospital departments of radiology must be administered by radiologists and not by hospital superintendents.

(2) Hospital radiologists must be assured incomes commensurate with their ability, comparable with those of colleagues in other fields of medicine and adequate to attract able young physicians into the field of radiology.

On these two issues we must stand without flinching and in doing so we will have the support of our patients, who are the public, our colleagues in other fields of medicine, hospital board members and trustees, and the responsible leaders in hospital administration.

950 East 59th St.
Chicago, Illinois

REFERENCE

1. HODGES, P. C. Development of diagnostic X-ray apparatus during the first fifty years. *Radiology*, 1945; *45*, 438–448.

Vol. 74, No. 6

IDIOPATHIC CHRONIC ULCERATIVE COLITIS AND REGIONAL ENTEROCOLITIS*

CLINICOPATHOLOGIC CORRELATION

By A. JAMES FRENCH, M.D., *and* S. A. VANDER, M.D.

ANN ARBOR, MICHIGAN

THE original description of the condition now known as idiopathic chronic ulcerative colitis probably is lost in medical antiquity, but Abercrombie[1] in 1830, Wilks and Moxon[30] in 1859, and White[29] in 1888 identified examples of such a disease and provided very adequate clinicopathologic data. The etiology and pathogenesis of the disease are not established, but Bargen,[3] Lium and Porter,[13] Felsen and Wolarsky,[6] Rowe,[18] and Warren and Sommers[25,26] have contributed significantly to these aspects of the condition.

Crohn, Ginzburg and Oppenheimer[5] separated regional ileitis (regional enterocolitis) from other inflammatory lesions of the intestines in 1932, and Crohn[4] reviewed his experiences with 298 cases of the disease in 1949. Experimental production of a comparable condition in dogs was reported by Reichert and Mathes,[17] while spontaneous disease of this type was found in dogs by Strande, Sommers, and Petrak.[23]

Kantor[9] in 1934 described the classical roentgenographic "string-sign" in regional (terminal) ileitis. An excellent diagrammatic representation of the theoretical pathogenesis of the disease was presented by Schepers,[19] while Hadfield,[8] Armitage and Wilson,[2] Rappaport, Burgoyne, and Smetana,[16] and Warren and Sommers[24] have reported detailed descriptions of pathologic changes in the disease.

MATERIAL

Surgical specimens obtained over a ten year period at the University of Michigan Hospital from 53 patients with idiopathic chronic ulcerative colitis and 30 patients with regional enterocolitis were reviewed clinically and pathologically.

TABLE I

		Ulcerative Colitis	Regional Enterocolitis
Sex	Male	34 (64.1%)	17 (56.6%)
	Female	19 (35.9%)	13 (43.4%)
Onset age	Average	25.5	31.8
First surgery, age	Average	32.6	36.0
Duration of symptoms	Average	7.1	4.2

The age distribution of the patients in both groups at the time of onset of clinical symptoms is shown in Figure 1. In addition to data relative to age incidence of the diseases, Table 1 includes the sex of the patients, age at first operation, and duration of symptoms before the first operation.

The apparent preponderance of males in the group of ulcerative colitis cases may be more relative than real inasmuch as twice as many males as females are admitted to University Hospital. Although the average age at onset of ulcerative colitis was six years earlier than that of regional enterocolitis, the symptoms were present three years longer before surgical intervention in the patients with ulcerative colitis than in those with regional enterocolitis. It must be appreciated that many so-called acute fulminant examples of ulcerative colitis and the early cases of regional enterocolitis would not be seen at an institution like the University of Michigan Hospital to which patients are admitted by referral, but would be cared for in their local community hospitals.

The data referable to religion, race, and occupation are presented in Table II. The

* From the Departments of Pathology and Surgery, University of Michigan Medical School, Ann Arbor, Michigan. Presented at the Fifty-fifth Annual Meeting, American Roentgen Ray Society, Washington, D. C., Sept. 21–24, 1954.

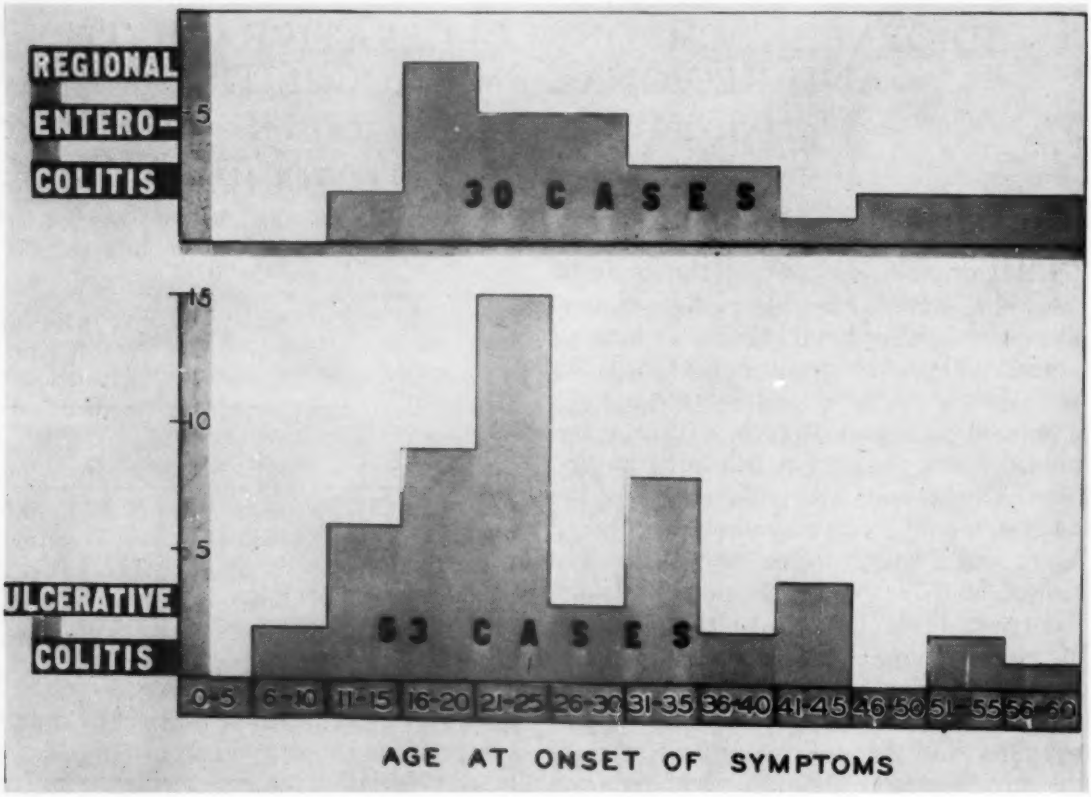

FIG. 1. Age distribution at the time of onset of clinical symptoms in 30 cases of regional enterocolitis and 53 cases of idiopathic chronic ulcerative colitis.

only conclusions to be drawn are those between these cases of ulcerative colitis and regional enterocolitis, as relative numbers of patients of a given race admitted to University Hospital are not included in this table. The clinical symptoms and signs are summarized in Tables III and IV.

The four most common symptoms in decreasing order of frequency noted in the patients with ulcerative colitis were diar-

TABLE II

		Ulcerative Colitis	Regional Enterocolitis
Religion	Protestant	36 (67.9%)	20 (66.6%)
	Jewish	5 (9.4%)	4 (13.3%)
	Other, or unknown	12 (22.7%)	6 (20.1%)
Race	Caucasian	52	28
	Negro	1	2
Occupation	Laborer	8 (15.0%)	6 (20.0%)
	"White collar"	29 (54.7%)	14 (46.6%)
	Housewife	16 (30.3%)	8 (26.7%)
	Undetermined		2 (6.7%)

TABLE III

Symptoms	Ulcerative Colitis		Regional Enterocolitis	
	No. of Cases	Percentage	No. of Cases	Percentage
Abdominal pain	38	71.7	29	96.7
Weight loss	31	58.4	12	22.6
Diarrhea	52	98.1	16	30.2
Nausea	0	0	4	13.2
Vomiting	1	1.9	7	23.3
Blood in stool	41	77.3	10	33.3
Fever & leukocytosis	9	17.0	2	6.6
Abdominal mass	1	1.9	2	6.6

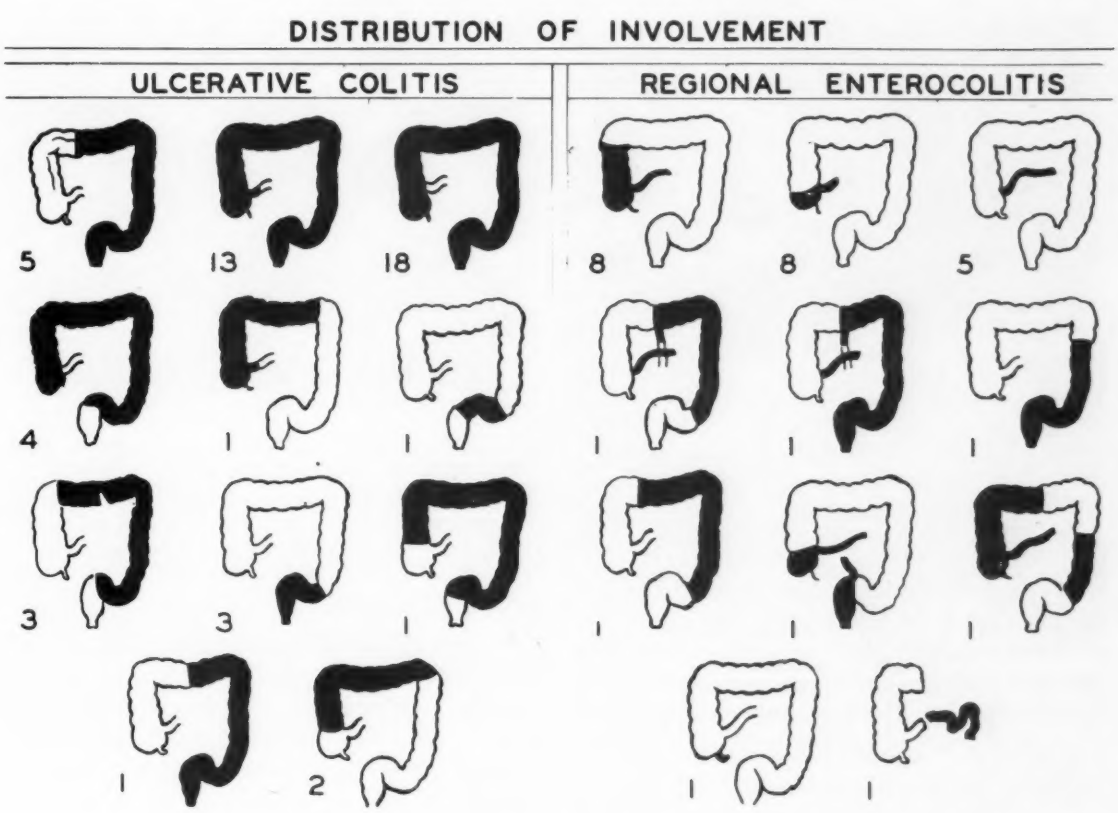

Fig. 2. Diagrammatic representation of the anatomic distribution of involvement in the two groups.

rhea, melena, abdominal pain, and weight loss, while those with regional enterocolitis complained of abdominal pain, melena, diarrhea, and vomiting.

In ulcerative colitis the five most common signs of the disease were malnutrition, abdominal tenderness, anal fistula, abdominal mass, and fever and leukocytosis. The patients with regional enterocolitis had malnutrition, abdominal tenderness, abdominal mass, and fever and leukocytosis in descending order of frequency of occurrence. Follow-up data are presented in Tables v to viii.

Table IV

Signs	Ulcerative Colitis		Regional Enterocolitis	
	No. of Cases	Percentage	No. of Cases	Percentage
Abdominal tenderness	18	34.0	16	53.3
Abdominal mass	6	11.3	7	23.3
Malnutrition	37	68.8	16	53.3
Fever & leukocytosis	6	11.3	6	20.0
Anal fistula	10	18.8	3	10.0
Abdominal fistula	2	3.7	3	10.0
None	10	18.8	2	6.6

Morphology

The anatomic distribution of the lesions is depicted in Figure 2. As would be anticipated, 40 of the 53 patients with ulcera-

Table V

Time of Follow-up

	Ulcerative Colitis	Regional Entercolitis
Range	0–12.0 Years	0–20.0 Years
Average	3.95 Years	4.8 Years
Mean	3.0 Years	4.0 Years

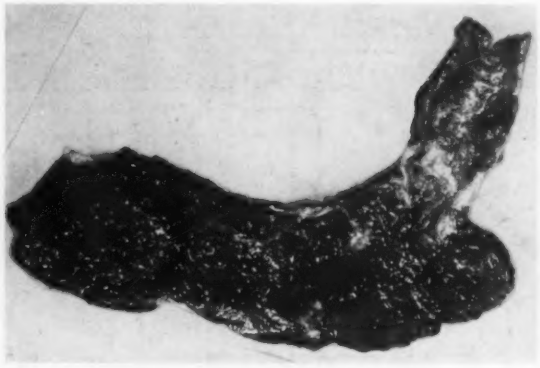

FIG. 3. Ileocecal area from a male, aged sixteen, with fulminant ulcerative colitis, perforation of the cecum, acute fibrinopurulent peritonitis, and death.

FIG. 4. Part of colon and ileum of a male, aged twenty-three, with a history of diarrhea and abdominal pain for one and one-half years. Active chronic ulcerative colitis and ileitis. Small ulcers and minute granulomatous pseudopolyps.

tive colitis had rectal involvement, with or without more widespread colonic changes. The terminal ileum was affected in 14 instances, or about one-fourth of the patients, while right colonic involvement alone or with ileal involvement was present in 18 instances. The appendix was involved in most patients with cecal disease, and in one case the location of the process was not well described and hence was not included in the illustration.

The patients with regional enterocolitis had ileal involvement alone in 5 instances, jejunal involvement in one, and colonic change only in 2 instances. Small intestinal involvement was present in 28 patients, and in one case the anatomic descriptions were inadequate for exact localization and inclusion in the figure.

Gross pathologic changes in chronic ulcerative colitis included focal or diffuse colonic, appendiceal, or distal ileal ulcers with little inflammatory reaction below the submucosa. The ulcers were often small and inconspicuous grossly, but obvious microscopically. No regular distribution of ulcers was found such as that described over the taeniae.[13] No specific microscopic char-

TABLE VI

CONDITION OF PATIENT AT TIME OF FOLLOW-UP

	Ulcerative Colitis		Regional Enterocolitis	
Alive				
Excellent	17	32.7%	10	33.3%
Fair	20	37.0%	7	23.4%
Poor	3	5.7%	5	16.7%
	40	75.4%	22	73.4%
Dead	10	18.9%	4	73.4%
Not located in this survey	3	5.7%	4	13.3%
	53	100.0%	30	100.0%

TABLE VII

DEATHS

Ulcerative Colitis (9 patients)		Regional Enterocolitis (4 patients)	
Cause	Post-operative	Cause	Post-operative
Unknown	5 years	Peritonitis	14 days
Peritonitis	4 days	Cancer, cervix	3 years
Peritonitis	10 days	Volvulus	1 year
Intestinal obstruction	2 years	Peritonitis	3 weeks
Colitis	1 year		
Colitis	1 year		
Colitis	19 days		
Cancer, ovary	2 years		
Acute yellow atrophy	3 months		

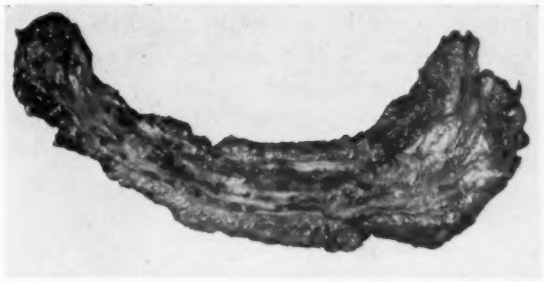

Fig. 5. Chronic ulcerative colitis. Left portion of colon from a female, aged thirty-nine, who succumbed to homologous serum hepatitis following partial colectomy.

acteristics of the ulcers were found, although the inflammatory reaction was exudative and not associated in uncomplicated cases with fibrosis and granulomatous change other than vascular granulation tissue. Mesenteric lymph node change was not prominent other than slight non

Table VIII

CONDITION OF PATIENTS LIVING WITH ULCERATIVE COLITIS

	Ileostomy	Colostomy	Neither
Excellent	14—82.3%	0—0%	3—17.7%
Fair	14—70.0%	1—5%	5—25.0%
Poor	2—66.7%	0—0%	1—33.3%

Fig. 6. Pseudopolyposis of the mucosa of the colon in chronic ulcerative colitis. Marked edema of the submucosa. (×20)

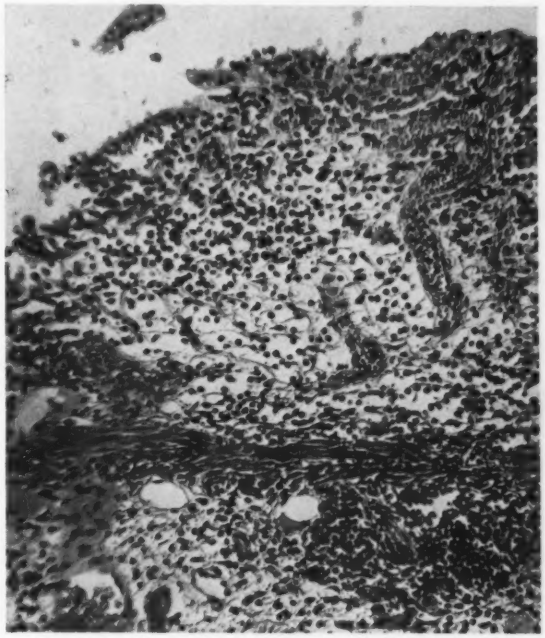

Fig. 7. Vascular pyogenic granulation tissue forming a pseudopolyp in an ulcerated area shown in Figure 6. Intact muscularis mucosae. (×120)

specific lymphadenitis. No thrombi were found so that the designation thrombo-ulcerative colitis may be inappropriate.

In regional enterocolitis the ileum, other parts of the small intestine and/or the appendix, and the colon grossly were characterized by cicatrizing change or productive inflammation with narrowing of the lumen of the involved area by fibrosis and

Fig. 8. Ulcerated, mainly epithelial, pseudopolyp in ulcerative colitis. (×20)

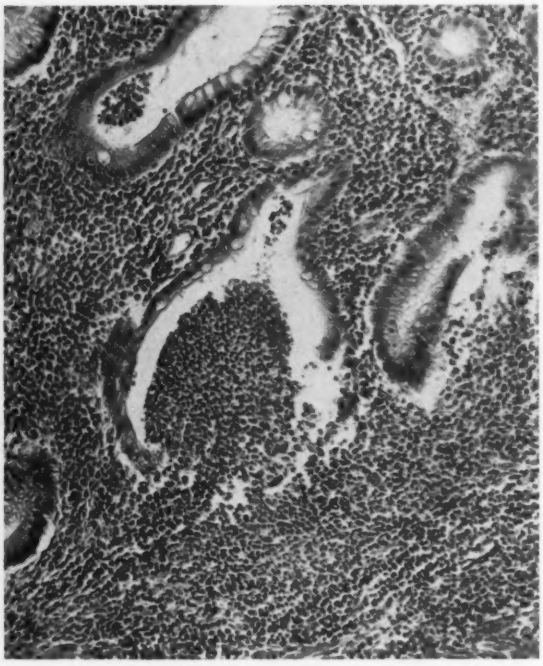

FIG. 9. Crypt abscess in a pseudopolyp in chronic ulcerative colitis. Hyperplasia of colonic mucosa with increased production of mucin. Inflammatory cells include lymphocytes and plasma cells in addition to neutrophils. (×120)

granulomatous inflammation of the wall. Pseudotubercles were present in the intestine and mesenteric or retroperitoneal lymph nodes in less than one-half of the cases studied. The tubercles contained less epithelioid cells and usually showed no caseous necrosis, in contrast to tubercles in true tuberculosis. The giant cells often contained vacuoles. Lymphatic stasis was not prominent, and when present could have been the effect rather than the cause of the inflammatory change.

The exudative inflammatory process which characterizes idiopathic chronic ulcerative colitis is depicted grossly in Figures 3, 4 and 5, and microscopically in Figures 6–11. Pseudotubercles are illustrated in ulcerative colitis in Figures 12 and 13. A barium granuloma in a case of ulcerative colitis is shown in Figures 14 and 15.

Adenocarcinoma mucosum (Fig. 16 and 17) developed in 2 of the 53 cases of ulcerative colitis, or 3.8 per cent. This is comparable to the incidence given by Weckesser and Chinn[28] and by Kiefer, Eytinge, and Johnson.[11] Both cases had had ulcerative colitis more than ten years. Skir[21] stated that a period of ten years or longer occurred before carcinoma developed in fistulas from other diseases.

Regional enterocolitis is characterized

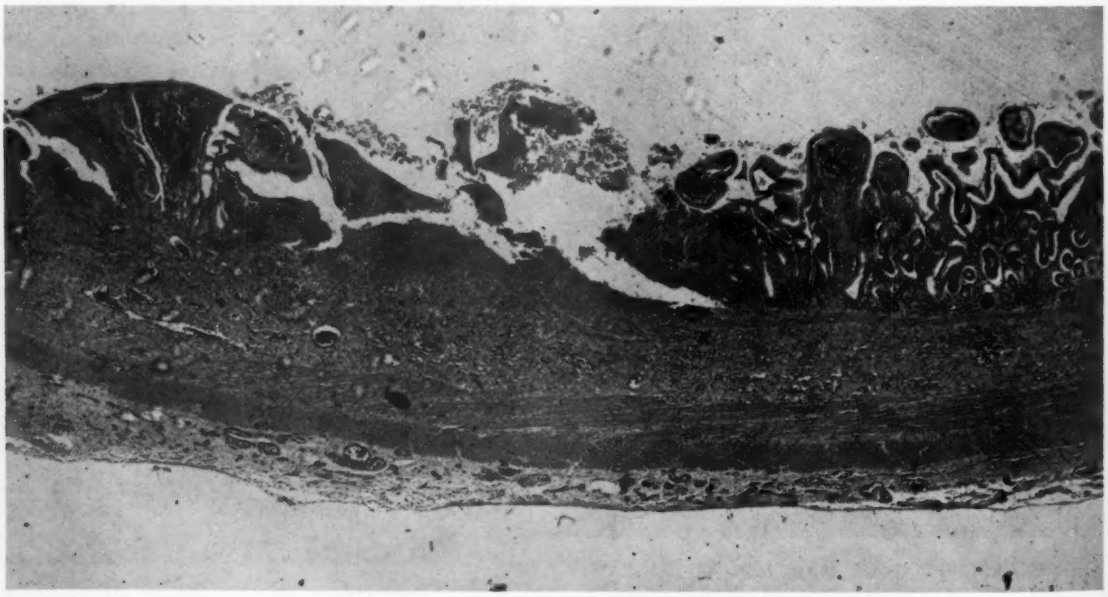

FIG. 10. Ulceration of the terminal portion of the ileum in chronic ulcerative colitis with ileitis. (×20)

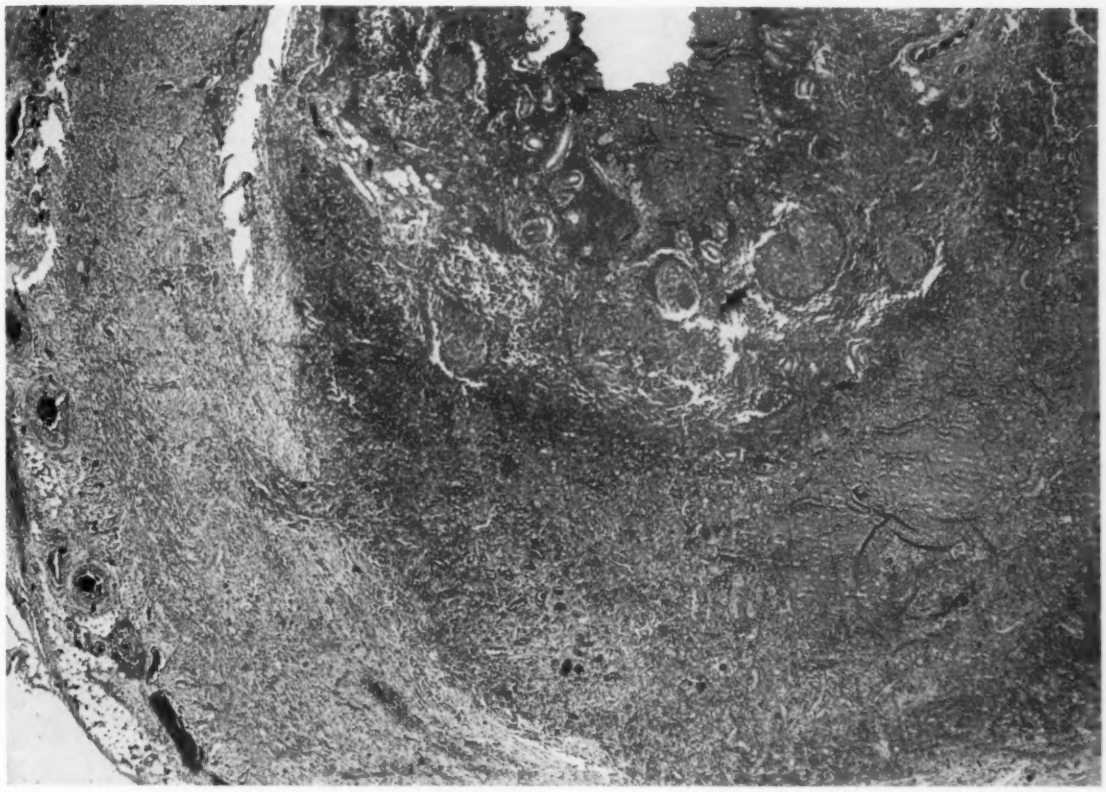

Fig. 11. Appendiceal involvement in chronic ulcerative colitis. (×20)

by productive inflammatory changes. Gross changes are illustrated in Figure 18. Microscopic features are shown in Figures 19–25. Less than one-half of the specimens contained the tubercles or pseudotubercles (Fig. 19–25) that are found in classical examples of the disease. Brunner's type glands are illustrated in colonic mucosa adjacent to an area of ulceration in Figure 25.

DISCUSSION

Most inflammatory lesions of the intestines have been separated into specific groups by clinical, etiologic, roentgeno-

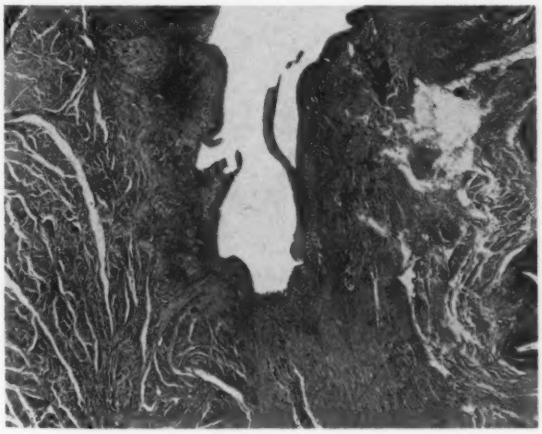

Fig. 12. Pseudotubercles in an anal fistula in chronic ulcerative colitis. (×20)

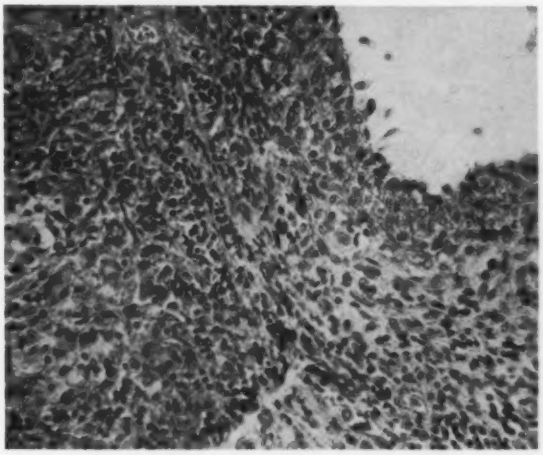

Fig. 13. Epithelioid cells and pseudotubercles in the wall of an anal fistula. (×120)

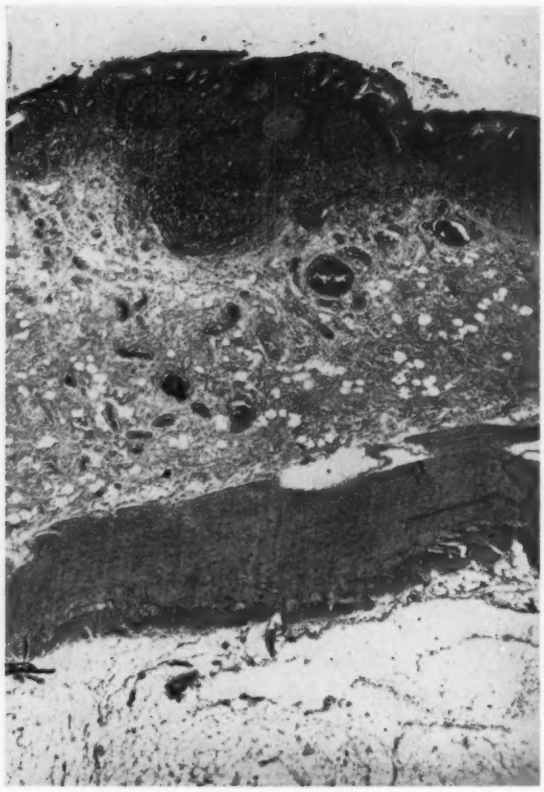

FIG. 14. Barium sulfate granuloma in the colon in chronic ulcerative colitis. (×20)

graphic, and pathologic differences. Idiopathic chronic ulcerative colitis and regional enterocolitis are, as a rule, separable from each other and from other intestinal

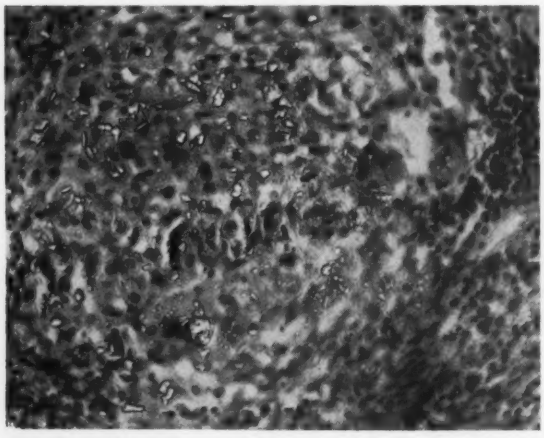

FIG. 15. Barium granuloma with crystals and epithelioid cells in the submucosa of the colon in chronic ulcerative colitis. (×120).

FIG. 16. Adenocarcinoma mucosum arising in the colon in chronic ulcerative colitis. (×20)

inflammatory processes,[24] but while the typical cases are readily distinguishable, there is a group of cases that are indistinguishable. It is possible that, at least in

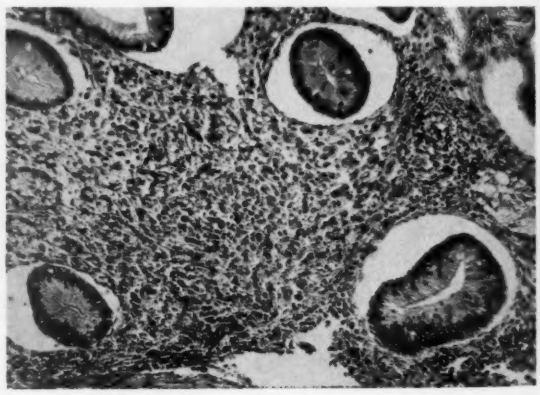

FIG. 17. A less well differentiated area of the same neoplasm. (×120)

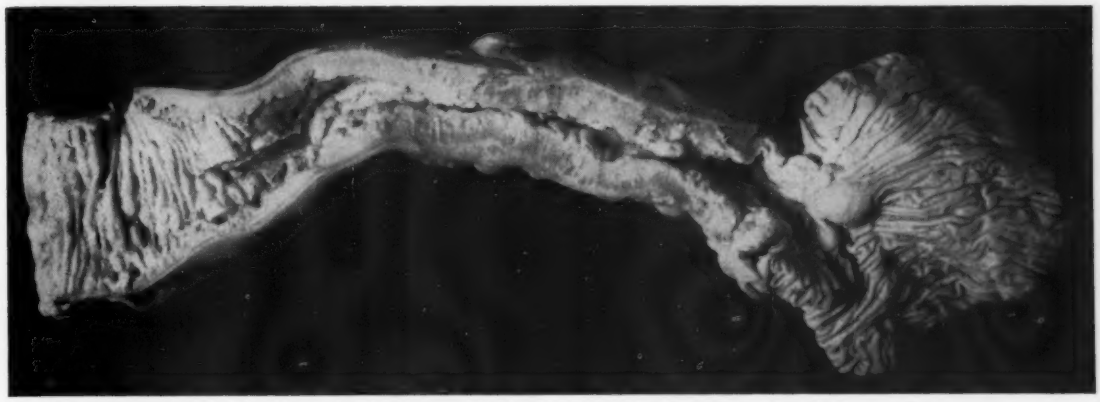

FIG. 18. Terminal ileum from a female, aged thirty-two, with regional ileitis. History of episodes of diarrhea and lower abdominal pain for eight years.

some instances, one of the conditions may evolve finally as the other, or that both are related.[6] Certainly, some examples require combined clinicopathologic and roentgenologic study for proper classification.

Tubercles or pseudotubercles may be present in the granulation tissue of the intestinal lesions or in the regional lymph nodes in either disease, but are much more frequently encountered in regional enterocolitis. Schaumann bodies or calcospherites were present in giant cells in the granulation tissue of two examples of regional enterocolitis. Necrosis was found in the tubercles in one example of regional enterocolitis. No evidences of generalized sarcoidosis[27] were found in specimens which contained tubercles.

Metaplastic change in ileal or colonic epithelium occurred in two examples of regional enteritis and in one of ulcerative colitis. The intestinal glands at the margins of ulcerated areas closely resembled Brunner's glands of the pylorus and duodenum. Liber[12] and Stout[22] have stressed similar changes in the intestine and stomach, respectively.

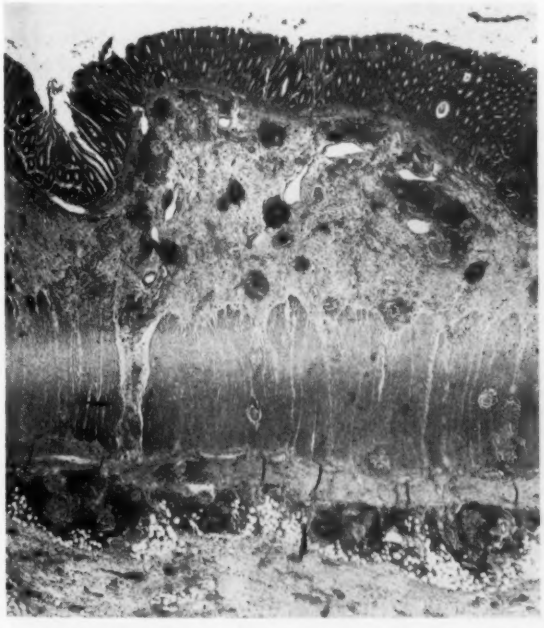

FIG. 19. Pseudotubercles throughout the wall of the colon and adjacent to adipose tissue. Regional colitis. (×20)

FIG. 20. Granulomatous focus in the submucosa of the colon in regional colitis. (×120)

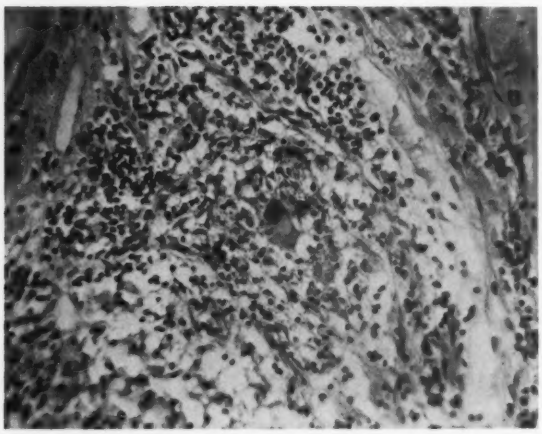

FIG. 21. Pseudotubercle with little epithelioid cell reaction and a somewhat pyknotic, constricted-appearing, multinucleated giant cell. (×120)

Barium sulfate crystals were found in two specimens from patients with ulcerative colitis, and the resulting granulomas were comparable to those described by Kay.[10]

While various bacteria, viruses, trauma, effects of enzymes such as lysozyme, excess psychic trauma, and allergy[18] have been postulated as etiologic factors in ulcerative colitis, and in part in regional enterocolitis, no agreement has been reached. Netter has diagrammatically represented the possible relationships of allergy to diseases of these types as presented by Silbert.[20] Certainly tissue hypersensitivity might explain the chronicity of these processes, but the initiating factor would be more difficult to envision.

Paulley[15] related the psyche to ulcerative colitis, but Ginsberg and Ivy[7] failed to see any such relationship, and Bargen[3] stated, "Is it not true that all chronic human diseases are inclined to be associated with disturbances of the psyche? Anxiety and emotional upsets do not cause ulcerative colitis, but its progression and failure to respond to treatment may well be aided and abetted by an unhappy frame of mind."

CONCLUSIONS

1. Fifty-three cases of idiopathic chronic ulcerative colitis and 30 cases of regional enterocolitis are reviewed from the surgical material during a ten year period at the University of Michigan Hospital.

2. No significant sex difference was found in the two conditions.

3. The average age of onset was six years less in ulcerative colitis than in regional enterocolitis.

4. Most cases of ulcerative colitis involved the colon alone, but the ileum was involved in 14 cases, and the colon other than the rectum in 12 cases.

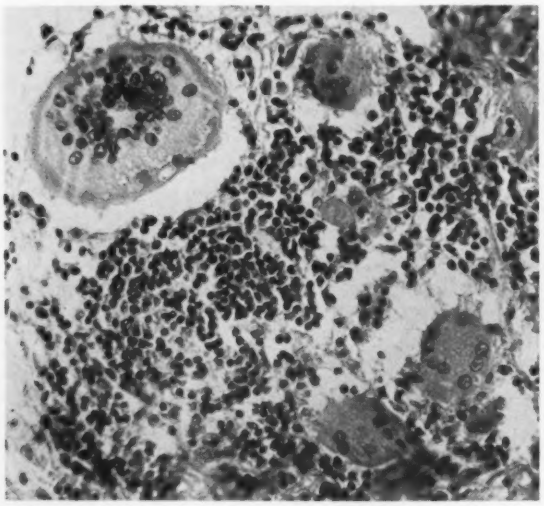

FIG. 22. Giant cells in a pseudotubercle in regional enterocolitis. (×240)

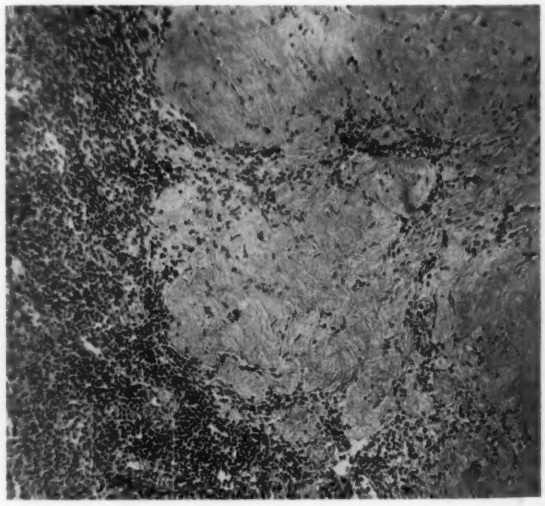

FIG. 23. Old hyalinized pseudotubercles in mesenteric lymph node in regional enterocolitis. (×120)

Fig. 24. Appendiceal involvement in regional enterocolitis. (×20)

Fig. 25. Brunner's type glands adjacent to an ulcer of the colon in regional enterocolitis. (×20)

5. The exudative inflammatory process seen in idiopathic chronic ulcerative colitis and the productive inflammation which characterizes regional enterocolitis usually permit separation of these conditions from each other and from other inflammatory processes in the intestines.

6. Idiopathic chronic ulcerative colitis and regional enterocolitis are clinicopathologic and roentgenologic entities which in most instances are readily distinguishable.

7. The etiology and pathogenesis of these conditions are not established, but tissue hypersensitivity reactions may account for the chronicity of the processes.

8. Pseudotubercle formation, metaplasia of intestinal epithelium to Brunner's type glands, and barium granulomas may be encountered in either disease.

9. Carcinomatous change occurred in 3.8 per cent of the cases of ulcerative colitis, and is a complication to be anticipated in long-standing cases.

10. Carcinoma was not found to have developed in regional enterocolitis in our material and was not noted in the literature reviewed.

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INFLAMMATORY LESIONS OF THE ESOPHAGUS AND STOMACH*

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EXCLUDING the ordinary expressions of peptic ulceration, inflammatory lesions of the esophagus and stomach are infrequently encountered during the course of roentgenologic examination of the upper gastrointestinal tract. In sharp contrast, esophagoscopy and gastroscopy appear to yield numerous evidences of abnormality involving the mucosal surfaces of these organs which are explained on the basis of inflammatory disease. The basic dissimilarity of direct visual and indirect roentgenologic procedures is the obvious explanation for this wide disparity between observations, even within the same clinical material. Deviations from the accepted normal, which consist of alterations in color and minute surface patterns, are certainly well within the range of visible detection when the esophagoscope and the gastroscope are employed, whereas such expressions of abnormality produce no detectable roentgenologic signs. It is only when pathologic processes alter the size and shape of the lumen, the active and passive movements of the walls, and the transit of ingested materials that any pathologic process involving these hollow viscera becomes apparent to the radiologist. He is relatively blind to superficial surface appearance, whereas the endoscopist is at great disadvantage when disease processes involve more deep seated structures.

During the five calendar years, 1949–1953 inclusive, 21,008 roentgenologic examinations of the upper gastrointestinal tract were carried out at the University Hospital. In 62 per cent of these examinations no roentgenologic evidence of abnormality of any sort was discovered. The incidence of tangible evidences of esophagitis and gastritis, as recognizable by roentgenologic methods, is extremely low. The rarity of these conditions is best expressed by the observation that during this same five year period, among patients in whom roentgenologic examination was employed, only 93 final diagnoses of esophagitis and 101 of chronic hypertrophic gastritis are listed in the hospital's records. Roentgenologic interest in the purely inflammatory lesions of the esophagus and stomach stems not from their frequency but rather from the difficulty that is encountered in the recognition and the correct interpretation of the alterations in appearance which they produce.

Of the 93 patients reported to have proved esophagitis, specific underlying causes of one sort or another were recognizable in 51, whereas in 45 no causative factor could be proved. Review of the recorded roentgenologic observations in the 45 examples of non-specific inflammatory disease of the esophagus shows that stricture of 1–3 cm. in length was observed in 29 instances and more generalized narrowing, 3–15 cm., in an additional 12. While diminution of lumen width was the outstanding and arresting roentgenologic finding, irregularity of contour, demonstrable ulceration, associated mass and perforation were also recorded. In three-fourths of these patients the lesion was restricted to the lower third of the esophagus. Hiatus hernia, almost uniformly associated with visible shortening of the esophagus, was clearly demonstrated in 22 of the 45 patients, indicating that protrusion of a portion of the upper end of the stomach into the thoracic cavity may well be a common and important causative agent in the development of esophagitis. Giuseffi, Grindlay

* From the Department of Radiology, University of Michigan, Ann Arbor, Michigan. Presented at the Fifty-fifth Annual Meeting American Roentgen Ray Society, Washington, D. C., Sept. 21–24, 1954.

FIG. 1. 555875. Hiatus hernia with stricture, shortening and ulcer.

FIG. 2. 681608. Hiatus hernia with stricture, shortening and ulcer.

and Schmidt[2] report interesting observations following the experimental production of esophageal hiatal hernia in dogs. They found that when sliding hiatal hernias were produced surgically, 90 per cent of the dogs upon whom the operation was performed developed mild to severe eso-

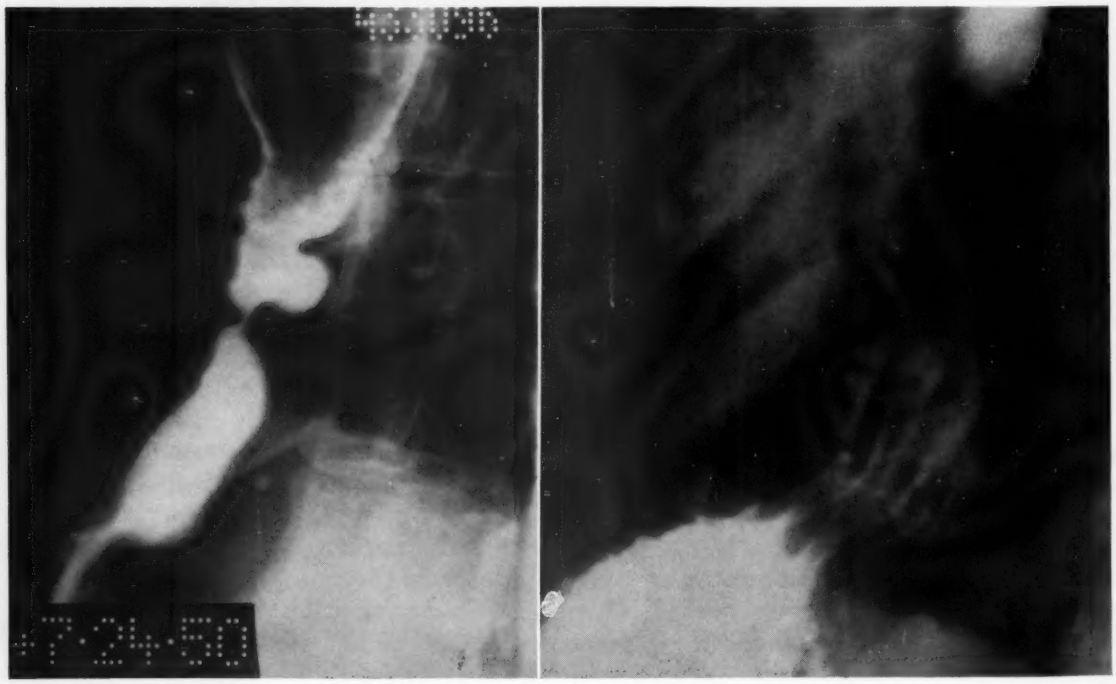

FIG. 3. 433096. Hiatus hernia with stricture, shortening and ulcer.

phagitis within 8–16 weeks. Friedenwald and associates[1] found that they could not produce ulceration in normal dogs by bathing the esophageal mucosa for long periods with 10 per cent hydrochloric acid. When, however, the mucosa was first traumatized with biting forceps, repeated washings with 10 per cent hydrochloric acid produced rather typical chronic ulceration. As observed in human material, the esophagitis presumably associated with hiatal hernia will not always be encountered at the stage when frank ulceration can be demonstrated. More often recognition of the situation must depend upon demonstration of the hernia and associated esophageal shortening with some degree of luminal narrowing (Fig. 1, 2, 3, 4 and 5).

It is apparent that not all gastric herniations through the esophageal hiatus produce recognizable signs of esophagitis for in the course of any considerable series of gastrointestinal examinations the finding of some degree of such herniation is relatively common (2.4 per cent in our five year series). This experience coincides with

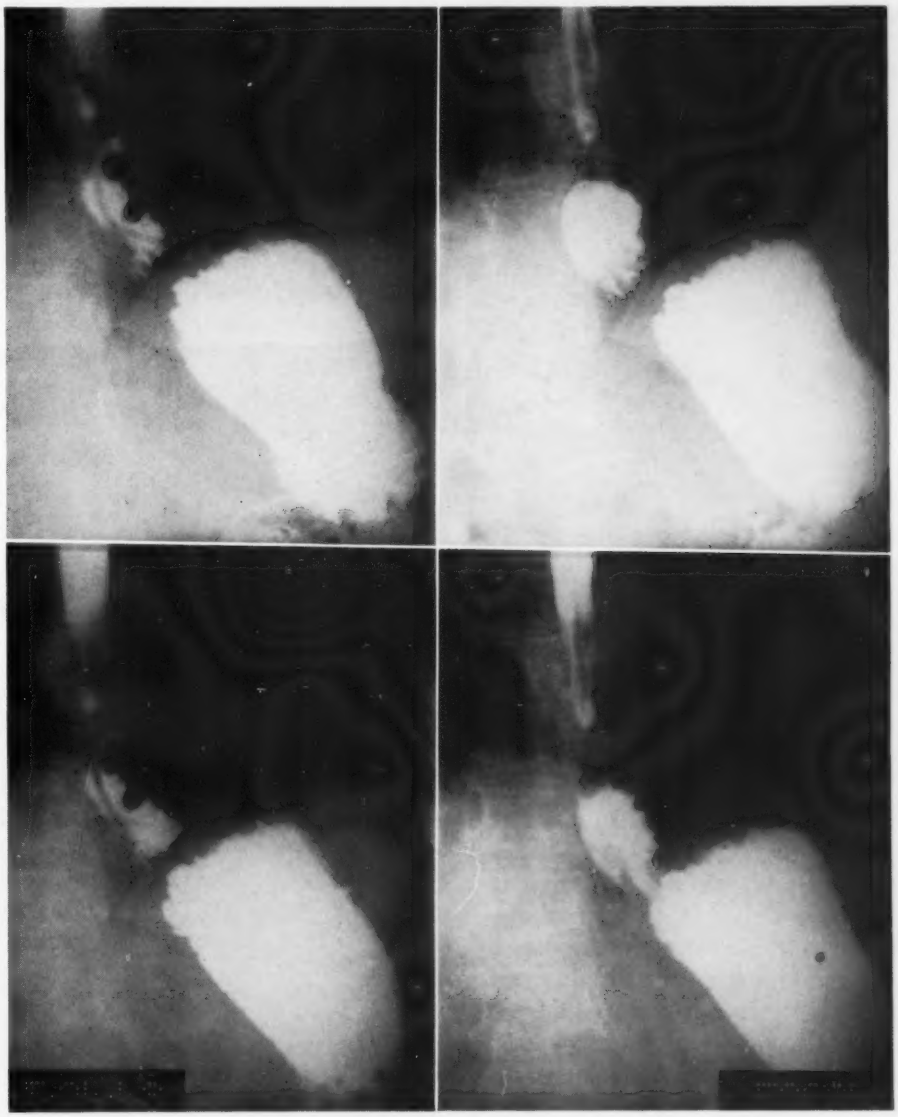

Fig. 4. 526840. Hiatus hernia (later resected) with stricture, shortening and ulcer.

FIG. 5. 685966. Hiatus hernia. Proved to harbor primary gastric carcinoma.

the experimental results of Giuseffi and his associates, who report that esophagitis occurred in but 55 per cent of those animals in whom an anchored, partially thoracic stomach was produced surgically, whereas the incidence was nearly twice as high when sliding hernias were produced. It would seem to be important for examiners at the fluoroscope to make every effort to identify observed hiatal hernias as fixed or of the sliding type. The roentgenologic demonstration of peptic ulceration involving the esophagus is less dependable than the results of endoscopic examination because by the latter method extremely shallow mucosal erosions, invisible by the techniques of roentgenology, can be demonstrated.

Among the 51 patients with inflammatory lesions of the esophagus of frankly specific character, chemical burns following the ingestion of excoriating substances, either by accident or with suicidal intent, numbered 35 or 68 per cent. Here, as in the nonspecific group, visible narrowing of the lumen, sometimes of profound degree, is the outstanding roentgenologic sign (Fig. 6, 7 and 8). The web-like band of the Plummer-Vinson syndrome, though uncommon in our experience, is ordinarily clear cut when observed. The sharp, annular constriction at or near the esophagogastric junction described by Schatzki[4] is also very obvious when present (Fig. 9 and 10). Irregularity of wall and sometimes ulceration has been observed in some instances of cardiospasm and also in association with varices of long standing (Fig. 11). Rare and most interesting examples of non-neoplastic esophageal disease are represented in this patient group by single instances of pemphigus (Fig. 12) and scleroderma (Fig. 13). Occasionally, following heavy doses of irradiation directed

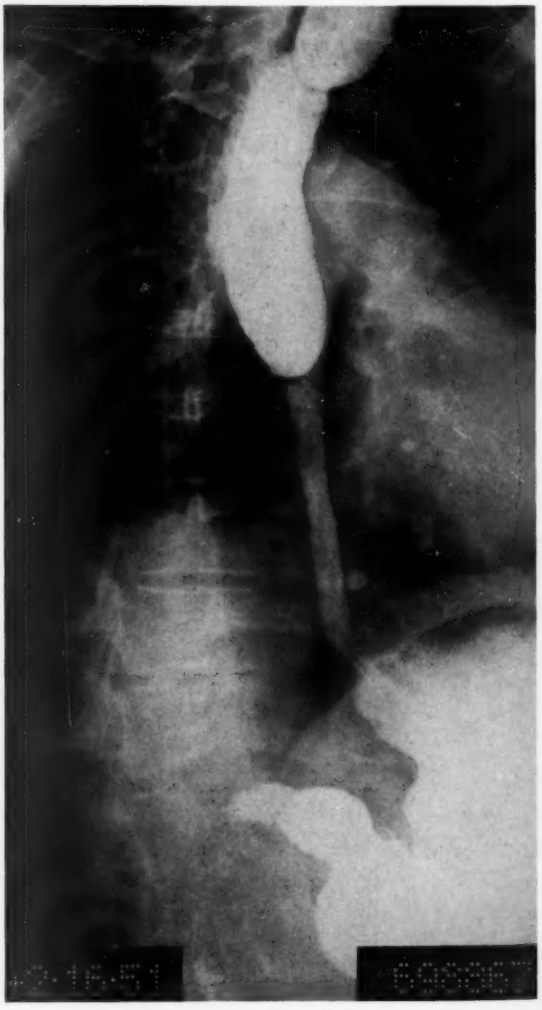

FIG. 6. 698867. Lye stricture.

toward the mediastinum, permanent alteration of the lumen in the walls of the esophagus can be demonstrated (Fig. 14). It is worthy of note that both by roentgenologic and endoscopic examination lesions ultimately proved to be purely inflammatory in nature were thought to present evidence of primary neoplasm in four instances, later to be disproved by tissue examination.

Exclusive of peptic ulcer, confirmed instances of inflammation of the gastric walls are encountered with infrequency and it is common to classify such material as acute or superficial gastritis, chronic hypertrophic gastritis and chronic atrophic gastritis. In the five years, 1949–1953 inclusive, 231 patients at the University Hospital were found to have one or another form of gastritis within this classification. The acute superficial and the chronic atrophic forms belong strictly in the field of the gastroscopist, whereas radiologists feel that their methods of approach can and do on occasion reflect the presence of the chronic hypertrophic form. Of 101 proved examples in this series, all of which were submitted to roentgenologic examination, positive findings were reported in 71, negative findings in 30. Gastroscopy was reported positive in 63 of the 101 cases.

Fig. 7. 693066. Post-intubation stricture.

Fig. 8. 684791. Multiple post-intubation strictures.

The roentgenologic diagnosis of chronic hypertrophic gastritis is based upon observations which are difficult to define and delimit. In general, as observed fluoroscopically, this disease imparts to the mucosal pattern of the stomach an obvious coarseness of the mucosal folds which attracts the attention of the observer (Fig. 15). Many factors operate to determine the breadth and the height of the rugae and it is the task of the observer to determine, if possible, whether transient contraction of gastric musculature is responsible or whether edematous or infiltra-

FIG. 9. 627760. Plummer-Vinson syndrome.

tive processes in the deeper layers of the stomach wall have produced the appearance. The differentiation is not simple. Palpation under fluoroscopic control offers the most satisfactory answer for the radiologist, for, if he is unable to flatten out the mucosal ridges under direct observation, the likelihood that the appearance is due to swollen, enlarged folds of abnormal mucosa is certainly great. When coarsened mucosal pattern gives way to normal appearances in the course of gastric filling and with the elapse of a few minutes of time, one can be readily convinced that the initial appearance carried no pathologic significance (Fig. 16 and 17). The radiologist is on sounder ground when mucosal overgrowth and coarseness is found in one segment only, although he may well be beset with worry as to whether the finding represents a sessile neoplasm or benign inflammatory disease (Fig. 18). The appearance of regional alteration of mucosa associated with frank gastric ulcer is so

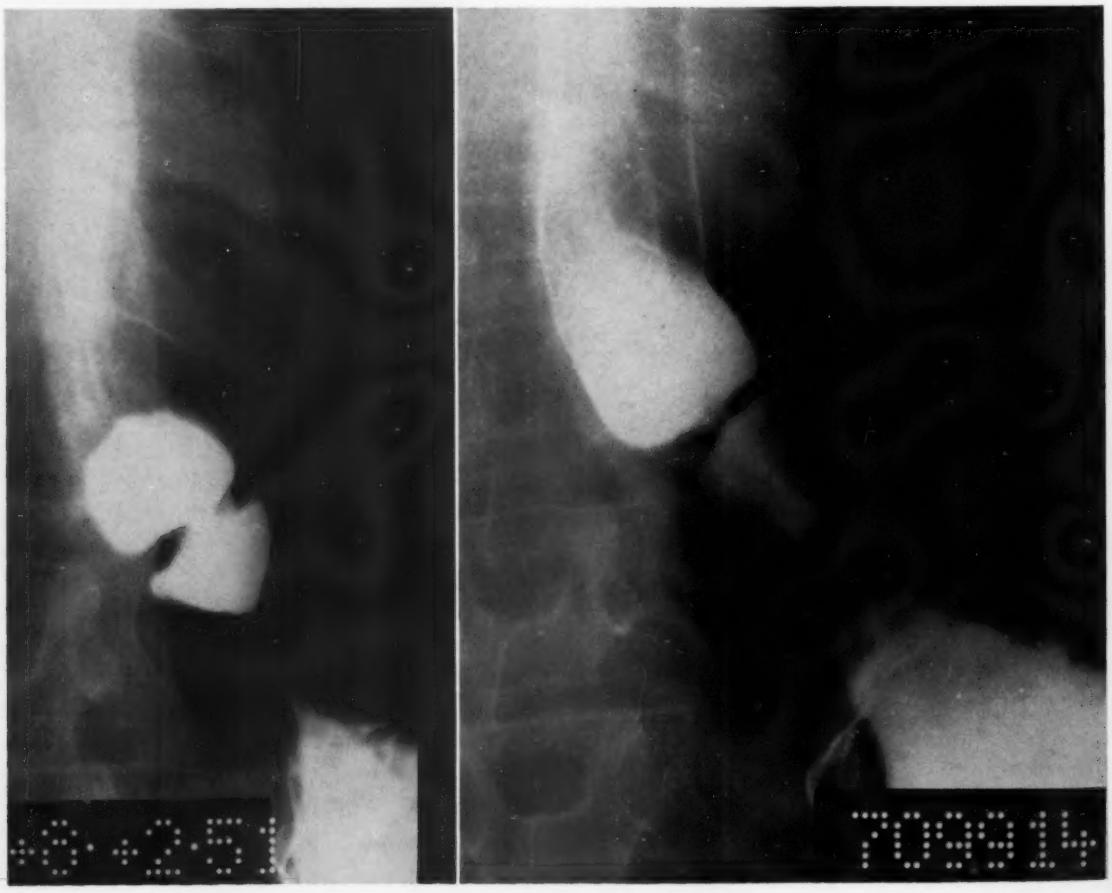

FIG. 10. 709914. Congenital band (Schatzki).

Fig. 11. 596514. Cardiospasm with associated esophageal ulcer. (No ulcer seen via esophagoscope.)

common in the experience of the radiologist that he expects it with great frequency and seldom includes in his report the observation of its presence. Certcertainly it is true that

Fig. 12. 639827. Pemphigus.

Fig. 13. 433015. Scleroderma. Dilatation, marginal deformity, depressed muscular activity.

Fig. 14. 606826. Postirradiation stricture (pulmonary carcinoma).

FIG. 15. 741964. Acute and subacute chemical gastritis.

FIG. 16. 460373. Generalized proliferative gastritis (surgical proof).

FIG. 17. 728184. Gastritis.

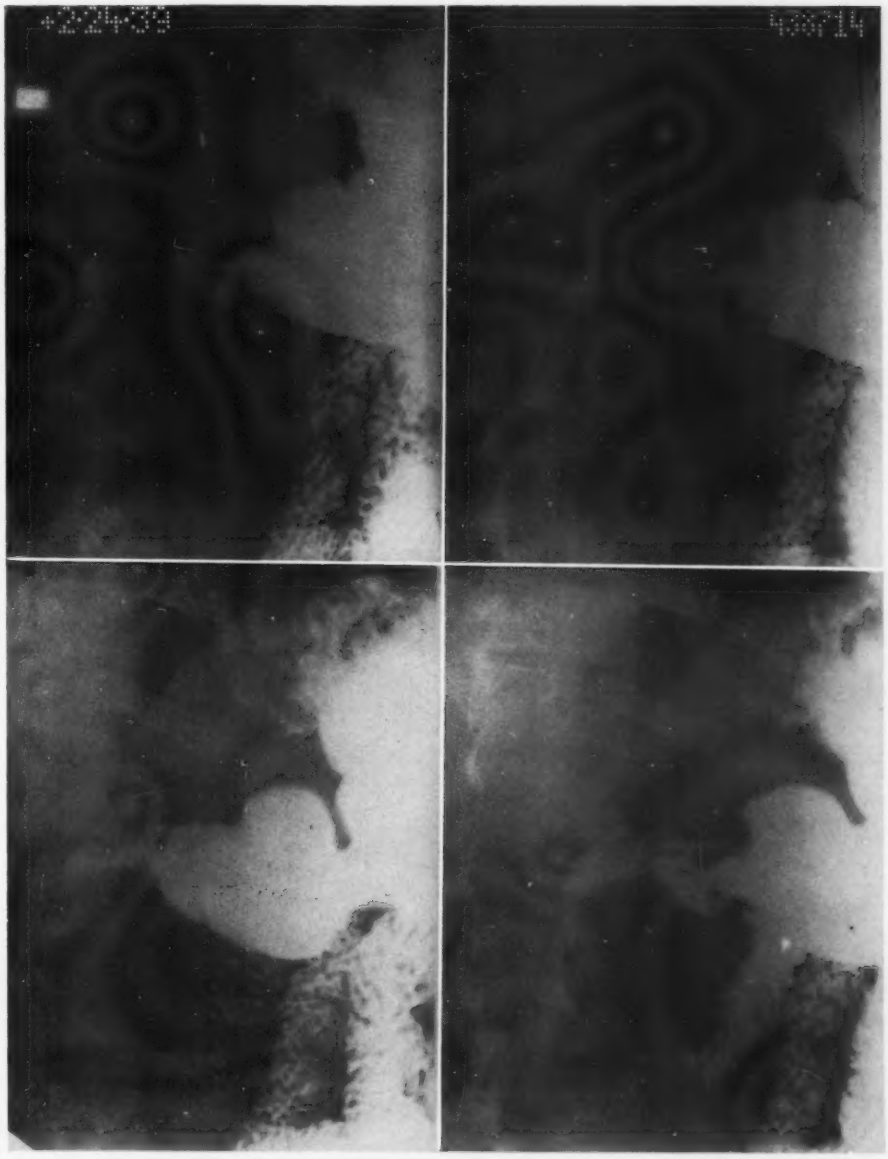

Fig. 18. 438714. Localized proliferative gastritis, gastric antrum.

the roentgenologic method is dependable only when hypertrophy of the gastric wall is well pronounced and this is in sharp contrast to the visual findings reported by the gastroscopist which, though they may be fully dependable and forthright, may be extremely superficial. Actually the language employed by the gastroscopist in describing his daily observations and those of the radiologist have led to a great deal of confusion and misunderstanding, and too often disagreement in both directions adds to this confusion.

There is one expression of hypertrophic gastritis which is clearly and convincingly apparent to the radiologist. The lesion in question almost invariably involves the lowermost segment of the antrum immediately proximal to the pylorus for a distance of 3 to 10 cm. Here, in sharp contrast to the surface of the mucosa elsewhere within the organ, folds are high, broad and tortuous,

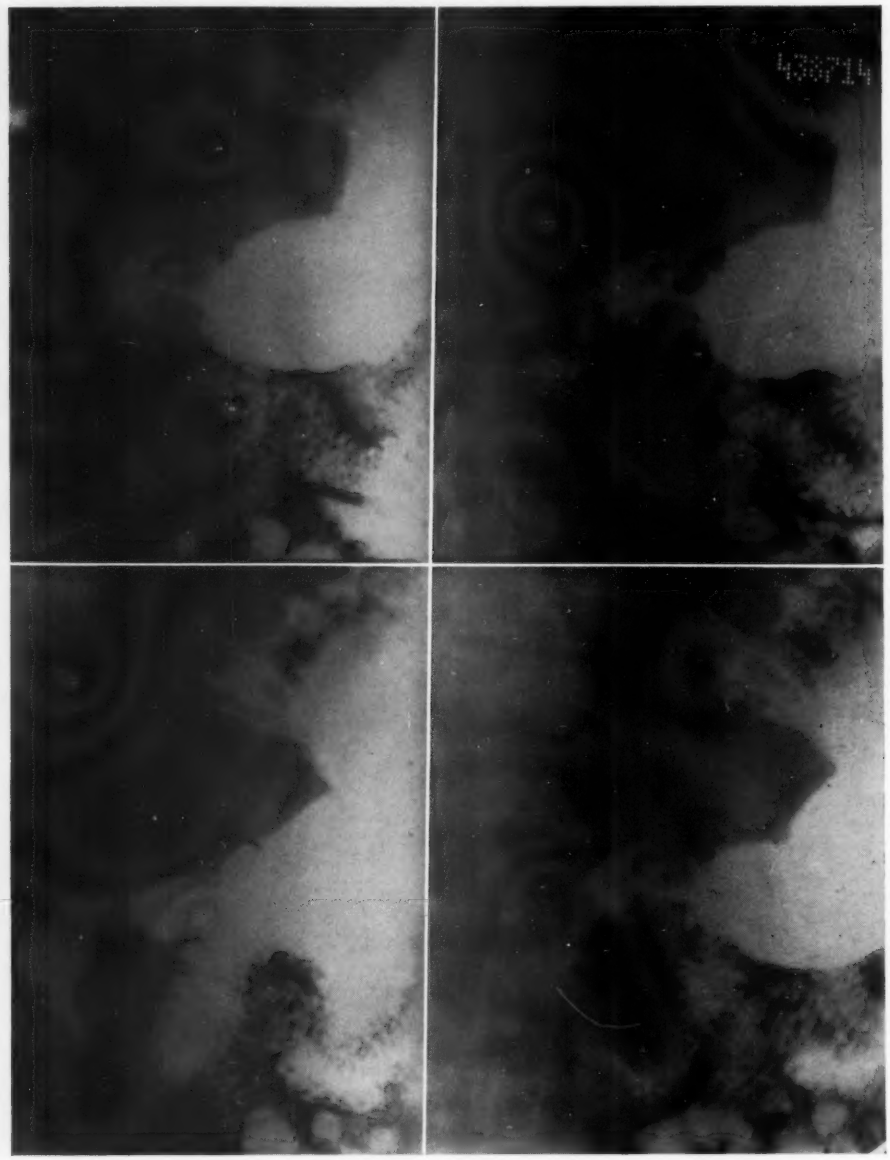

FIG. 19. 438714. Same patient one year later—surgical proof.

impossible of complete eradication even with forceful manual palpation. The irregularity is not confined to the mucosal surface alone for the lumen is ordinarily irregularly narrowed without the degree of associated rigidity encountered in neoplasm. It is common to find the base of the uninvolved duodenal bulb indented by the hypertrophic portion of the stomach and peristaltic activity, normally maintained at higher levels, is feeble or virtually absent through the deformed gastric segment. Ordinarily roentgenologic examination in patients with this condition yields evidences of clearly apparent abnormality which are difficult to differentiate from primary neoplasm, and it is commonplace to repeat the examination once or twice before the latter situation can be eliminated. Actually it is sound practice to recommend surgical exploration in all such cases because the radiologist can scarcely distinguish between them with complete honesty (Fig. 19). In this particular ex-

pression of hypertrophic gastritis it has been our experience that gastroscopic examination may likewise be ambiguous for the observer may never be able to introduce the instrument into the involved segment.

The abnormalities encountered throughout the small intestine and the colon, commonly spoken of as nonspecific regional enteritis, terminal ileitis, jejunitis, ileocolitis, and referred to frequently as Crohn's disease have not been encountered in our experience at the level of the stomach. Martin and Carr[3] have presented the case histories and findings in two young women as examples of Crohn's disease involving the stomach. Only one of these patients, the first, was substantiated by biopsy from the gastric wall, reported as follows: "The stroma is heavily infiltrated by lymphocytes, plasma cells and endothelial cells. Giant cells are present. No caseation or tubercle bacilli are found. A chronic granulomatous process like Crohn's disease appears to be present." We have been able to find no other published example of gastric disease suggestively related to the nonspecific inflammation of the small intestine.

The specific inflammatory processes of lues and tuberculosis, well authenticated with microscopic findings, have been reported although the incidence is extremely low. From time to time one does encounter generalized gastric polyposis but it has been our experience that the nodules recognizable on the mucosal surface on roentgenologic examination have always been true benign neoplasms rather than the inflammatory polyps of extremely small size described by the gastroscopist as "polypoid gastritis." Prolapse of gastric mucous membrane into the proximal duodenum by way of the pylorus can as a rule be identified without difficulty on the basis of roentgenologic examination. The presence of this state of affairs is presumptive evidence of associated gastritis involving the prolapsed portion, but in the eyes of the radiologist the mechanical displacement of redundant mucosa is the arresting and convincing sign.

SUMMARY

To the extent to which the methods employed in roentgenology are reliable in its detection, inflammatory disease occurring in the esophagus and in the stomach is a finding of relative infrequency. Acute and subsequently chronic sclerosing esophagitis following abnormal exposure to acid gastric contents in patients with sliding hiatal hernia is the most commonly recognized form of esophageal inflammatory disease. Luminal narrowing, whether short and high grade or more extensive and less complete, is the most arresting roentgenologic finding in esophagitis.

The bulk of confirmed examples of gastritis, as recognizable roentgenologically are of the chronic hypertrophic variety in which the deeper submucosal layers of the stomach are primarily and most extensively involved. The commonest expressions of hypertrophic gastritis as seen by the radiologist are of the localized or segmental variety, usually confined to the extreme lowermost portion of the gastric antrum. The roentgenologic differentiation between segmental hypertrophic gastritis and primary neoplasm is often difficult, sometimes impossible. Nonspecific regional inflammatory disease similar in character to lesions found with fair frequency in the small intestine and colon are certainly rarities in the stomach.

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ROENTGEN FINDINGS IN REGIONAL ENTERITIS*

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INFLAMMATORY lesions of the small intestine producing roentgenologic changes are being recognized with increasing frequency. By far, the most important of these, both in frequency and its clinical implications, is the entity which has come to be known as regional enteritis. As originally described in 1932,[5] this ailment was thought to affect solely the distal ileum. Numerous investigations subsequent to this have amply demonstrated that the intrinsic pathology of terminal ileitis may be seen in any portion of the small intestine, and, on rare occasions, even the stomach.[12,13,14,17]

Seven hundred and fifty patients with regional enteritis were studied over a period of one to twelve years and this occasioned the opportunity for serial clinical and roentgen investigations. Although many of the specific roentgen features of this disease have been described previously, this study will report the roentgen findings from the earliest alterations detectable roentgenologically to the late stenotic stage as well as the roentgen changes characteristic of the site of involvement. It is impossible to state whether earlier changes different from those described below exist or can be recognized as most of the patients are examined roentgenologically when the disease process is already well established. It has been possible, however, to show the early changes in recurrences following sidetracking operations and these changes are similar to those noted in the intestinal segments less severely involved by regional enteritis.

The symptomatology is dependent upon the stage of the disease, the site and the extent of involvement of the intestine. It is usually associated with a low-grade fever, loss of weight, abdominal cramps, diarrhea and, on occasion, incomplete obstruction. While complete obstruction almost never occurs in regional enteritis, symptoms are, on occasion, so threatening as to warrant surgical intervention. The diagnosis may be obscure for many months or years because the typical symptom complex either does not make its appearance at all, or does so at an advanced stage of the disease.

Physical examination may be entirely negative. The degree of nutrition roughly parallels the severity of the intestinal disease. An abdominal mass most frequently found in the right lower quadrant is occasionally present and the spleen may be enlarged. External fistulae to the anterior abdominal wall or flanks are also noted, as well as fistulous communications between the diseased intestine and the perirectal spaces, with the formation of perirectal abscesses and secondary proctitis. Clubbing of the fingers is occasionally observed.

PATHOLOGIC FINDINGS

Regional enteritis is a nonspecific granulomatous ulcerative, necrotizing, inflammatory disease of unknown etiology. Internal fistulae from the ileum to the adjacent viscera, external fistulae to the abdominal wall, and perirectal spaces are frequent. The significant pathological features that pertain to the roentgen findings will be included. The intestine is thickened, rigid, and hose-like with a blotchy red discoloration on its serosal aspect. In most cases the granulomatous process stops abruptly at the ileocecal value. However, extension as far as the descending colon has been reported.[2] The mesentery of the involved portion of small intestine is hugely thickened, soggy, edematous, and contains enlarged lymph nodes. Skip areas of normal bowel of varying lengths are characteristic.

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Matting of various intestinal loops to each other and to other structures, secondary to abscess formation or fistulae, may be noted.

On opening the bowel, the intestinal folds are distorted and broken up by the destructive ulcerative process, and rounded and blunted by edema, producing a coarsely nodular appearance which, in some cases, is sufficiently uniform to suggest a cobblestone pattern.[5] A series of small linear ulcerations on the mesenteric side of the bowel is almost always present. The thickening of the bowel is due primarily to an increase in the submucosa which is the seat of marked inflammatory hyperplastic changes. The muscular layer is also thickened, but to a lesser degree. Ulceration progresses and may be superficial or deep, with varying degrees of induration and cicatrization.

This may give rise to an inflammatory reaction in the submucosa muscularis and serosa, thus accounting for the abscesses and fistulae. The extent of the ulceration varies, leaving behind islands of inflamed mucosa with undermined edges. As cicatrization of the ulcerated areas takes place, the isolated islands of hypertrophic mucosa become prominent and assume a polypoid appearance. Later, there may be an attempt at healing of the ulcerated areas and the lumen will be lined with a layer of smooth, atrophic, glistening epithelium. When strictures are marked, the intervening segments of the bowel become considerably dilated. Microscopically,[9] the submucosal thickening is the earliest pathological process that occurs and is the result of edema and a widespread hyperplasia of lymphoid and reticulum cells. Giant cell tubercles resembling those seen in tuberculosis but without caseation are characteristic. Eventually, the nodules are obscured by more diffuse inflammatory infiltrations with numerous plasma cells and finally, by fibrous tissue.

ROENTGEN FINDINGS

Several investigators divide the roentgen findings in this disease into acute, subacute and chronic stages.[17] Since the classical form of regional enteritis is that of a low-grade inflammatory process, with episodes of acute exacerbation, it is difficult to identify such a clear-cut pattern. It is possible that acute granulomatous regional enteritis does occur, but the determination of the precise onset of this disease is almost impossible and what is described as acute regional enteritis may be the more active phase of this chronic illness.[1]

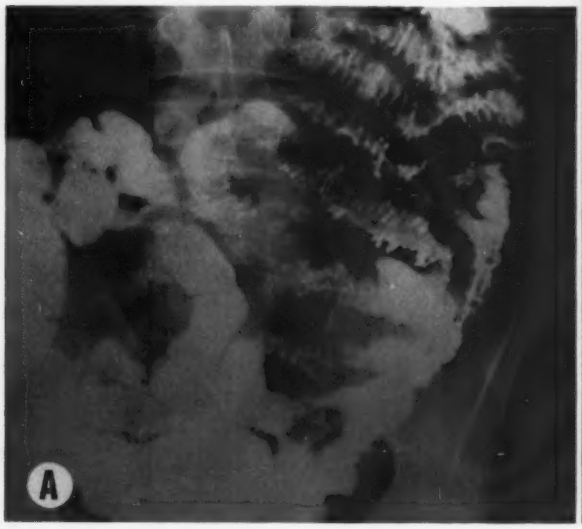
Proximal and distal extension of the disease process, despite repeated roentgen examinations extending over a period of many years, was not observed. The maximum length of involvement is determined on the initial roentgen studies. This is not true after exclusion operations when recurrent disease is frequent.

One of the prominent features of this disease is the development of stenosis with obstruction. Therefore, roentgenologically, the cases may be divided conveniently into stenotic and nonstenotic groups.[12,13] It is impossible to classify these groups as early and late, since the majority of cases may continue without stenosis for many years. Again, division into active and inactive seems inappropriate, as the patient with long segments of stenotic intestine may also exhibit considerable evidence of clinical activity, manifested by fever, diarrhea and abdominal pain.

Nonstenotic Phase. The roentgen findings closely parallel the pathologic features described in the previous paragraphs. Early changes are blunting, flattening, thickening and straightening of the valvulae conniventes. The folds are arranged in a fairly regular, symmetrical, parallel fashion, appear rigid and are perpendicular to the long axis of the intestine. The folds become thicker, irregular and partially fused (Fig. 1*A* and 1*B*). The lumen and contour become irregular. Occasionally the irregularity of the jejunal contour and valvulae is seen early without distinct blunting or thickening of the folds. These changes are due to the inflammatory submucosal and mucosal thickening. When ulceration occurs, a more characteristic

pattern is produced. Longitudinal streaks of barium recognizable as ulcerations appear (Fig. 2*A*). As the thick, blunted folds of mucosa are further destroyed, cobblestoning may be noted (Fig. 2*B*). This specific pattern is more common in the jejunum, probably because of the thicker valvulae in this region. Ulceration continues at the expense of the intervening islands of mucosa, replacing the cobblestone pattern by an irregular network of interlacing streaks of barium. The appearance at this stage has no uniformity or symmetry and is hazy and reticulated. Denudation of the mucosa is usually incomplete, leaving behind islands of inflamed mucosa which produce multiple smooth defects of varying size (Fig. 3*A*), Their prominence is increased by the narrowing of the bowel lumen, due to beginning cicatricial contraction which occurs at this stage. Finally, one may see the roentgenologic image of a uniform, rigid, cast-like tube filled with barium and presenting no mucous membrane pattern. This is similar to ulcerative colitis and represents the stage when scarring and regeneration of an atrophic mucosa is progressing (Fig. 3*B*). As scarring proceeds, the transition to the stenotic phase occurs. An uncommon but striking finding is the presence of large inflammatory polyps (Fig. 4*A*). Ordinarily the polyps are much smaller. Carcinomatous transformation of these polyps has not been observed.

Coincident with changes in the mucosa, other characteristic roentgen features occur. The bowel lumen reveals varying degrees of narrowing. Early the submucosal thickening and associated spasm are responsible for the narrowing of the lumen. Later, as fibrosis occurs, the narrowing is more marked and leads finally to the stenotic stage. In the early stages, rigidity of the contour and mucosal pattern is incomplete. Some flexibility or dynamic activity is evident in the change in contour and mucosal pattern during successive roentgenograms



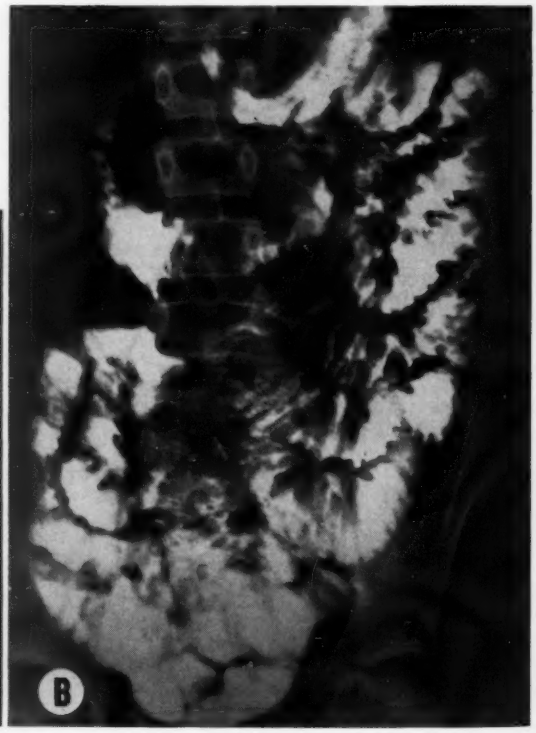


FIG. 1*A*. The mucosal folds are thickened, blunted and in some areas fused. The contour of the lumen is irregular. There is some rigidity and separation of the loops of intestine which also appear straightened or uncoiled. Diagnosis: Nonstenotic type of regional enteritis.

FIG. 1*B*. There is slight thickening and blunting of the folds with moderate irregularity of the contour of the bowel. Numerous small defects are noted which in some areas appear to be submucosal. There is slight rigidity of the loops of bowel. Diagnosis: Nonstenotic type of regional enteritis.

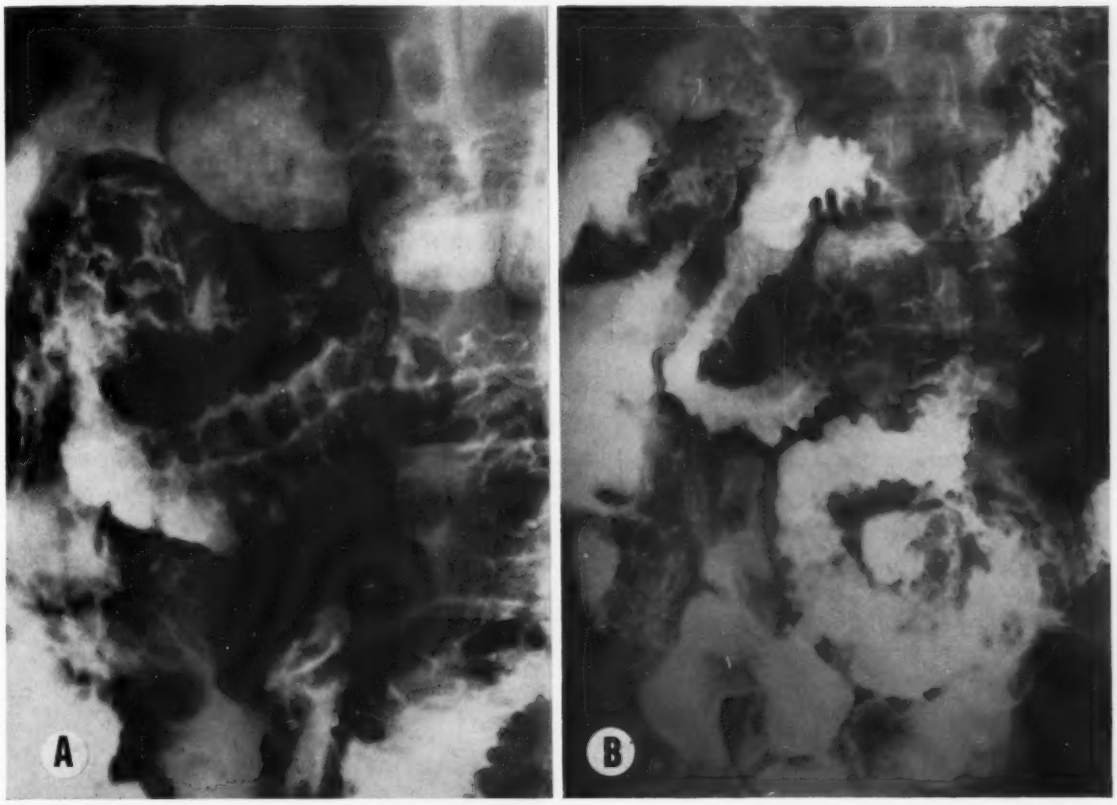


Fig. 2*A*. Longitudinal streaks of barium indicating ulceration are noted. The contour is irregular with some rigidity and separation of the loops of intestine. Diagnosis: Nonstenotic type of regional enteritis.

Fig. 2*B*. A cobblestone pattern is noted. In other areas the folds are thickened and blunted. There is moderate irregularity of the contour of the bowel. Diagnosis: Nonstenotic type of regional enteritis.

(Fig. 1*B*). Later, the roentgen appearance is fixed and unvarying (Fig. 3*A* and 3*B*). Pliability is lost. Also early in this disease, the normal serpentine or coiled pattern of the loops of small intestine disappears. The diseased segments appear to be straightened and rigid. This finding is probably due to loss of flexibility in the intestinal wall and mesentery, as well as longitudinal shortening due to spasm.

Not infrequently, the loops of intestine appear to surround a mass (Fig. 5*A*). Although this may be due to an abscess resulting from perforation, more often it is secondary to the indurated mesentery, associated with the marked increase in the mesenteric fat and the enlarged lymph nodes. The silhouette of the intestine may be hazy. This is due to the fact that the intestine is diffusely ulcerated, contains exudate and excessive secretions and the barium does not adhere to the walls. Despite the increase in intestinal content, the barium mixture remains fluid and homogeneous. Neither agglutination, clumping nor formation of masses of barium is found in regional enteritis as contrasted with the findings in the so-called sprue pattern. Segmentation such as is characteristic of sprue has not been observed. When unequivocal segmentation occurs, the diagnosis of regional enteritis should be suspect. Skip areas (Fig. 3*A*), that is, segments of normal intestine intervening between diseased segments, represent another characteristic feature. The length of a skip area may vary from a few inches to several feet. The extent of involvement on a roentgenogram may be quite accurately determined in most cases, since the transition of disease

to the normal intestine is fairly abrupt. An outpouching of the mucosa between the thickened folds (Fig. 4*B*), occurs in some cases, creating an appearance suggestive of large diverticula. This is peculiar in that it seems to affect one side of the intestine and not the other. These pseudodiverticula probably represent small eccentric skip areas.

Stenotic Phase. In the stenotic stage, many of the rigid loops described previously become constricted to a remarkable degree. These stenotic segments resemble rigid pipestems (Fig. 5*A*, 5*B*, 6*A* and 6*B*). This appearance is due to a marked thickening and contraction of the wall of the small intestine. The stenosis may extend through 1 or 2 cm., or over long segments. With severe narrowing dilatation of the proximal intestine may be marked. In many instances, it is difficult to state whether or not intrinsic disease is present in a dilated area. Very often disease is present when a loop of dilated intestine exists between two points of constriction. On the other hand, when there is a single area of constriction with proximal dilatation, disease may not be present in the dilated segment. However, because of retained secretions, secondary inflammatory changes, tension ulcers and muscular hypertrophy, the appearance of the dilated loops may be confused with the alterations seen in regional enteritis. This distinction is of great importance when considering surgical therapy. Many cases have not undergone operative intervention because the surgeon was of the opinion that the entire intestine was involved. Extreme dilatation of the intestine for long segments is rarely associated with intrinsic granulomatous disease.

Many of the roentgenographic phenomena observed in the nonstenotic phase of

FIG. 3*A*. There is rigidity and separation of the loops of intestine, with numerous small filling defects due to inflammatory polyps and eccentric skip areas. Diagnosis: Nonstenotic type of regional enteritis.

FIG. 3*B*. The distal jejunum and proximal ileum are involved. The mucosal pattern appears cast-like. Several small defects due to inflammatory polyps are noted. The loops of bowel are rigid and markedly separated. Diagnosis: Nonstenotic type of regional enteritis.

the disease are again noted in the stenotic phase. The mucosal pattern is usually reticulated or cast-like (Fig. 5*A*, 5*B*, 6*A* and 6*B*). Small filling defects, inflammatory polyps, irregularly distributed throughout the diseased segments, are noted. Skip areas and wide spacing between the segments of intestine are more striking.

The loops are rigid and maintain a constant position from film to film. The diseased segments of intestine seem to encircle an inflammatory mass. Fistulae, usually involving the distal ileum, may be seen. Occasionally, these are very difficult to demonstrate, especially when dilated loops of intestine produce overlapping. The fistulae may extend to and penetrate the abdominal wall.

In general the roentgen findings in regional enteritis are as described; however, there are certain characteristics which are peculiar to the area of involvement.

Ileum. The most frequent site of involvement of the small bowel with regional enteritis is the terminal ileum. Five hundred and six cases reveal involvement of the distal nine to twelve inches of terminal ileum. One hundred and twenty-six patients have involvement of the entire ileum.

The string sign which has come to be identified as the pathognomonic roentgen manifestation of regional enteritis is most frequently noted in this region. It has been described as a thin, linear shadow, suggesting a frayed cotton string in appearance (Fig. 7*A*). The cause of the string sign is incomplete filling due to irritability and spasm associated with marked ulceration and may be seen in both the nonstenotic and stenotic phases of this disease. Repeated spot films will demonstrate the fact that some distensibility is still present in this segment (Fig. 7*B*). The bowel proximal to the string sign may or may not be dilated depending on the stage of the disease. In the nonstenotic phase the proximal intestinal lumen is generally not dilated despite the marked narrowing associated with the string sign, indicating the importance of spasm in producing this characteristic appearance. This spasm is usually inconstant. When persistent, however, tem-

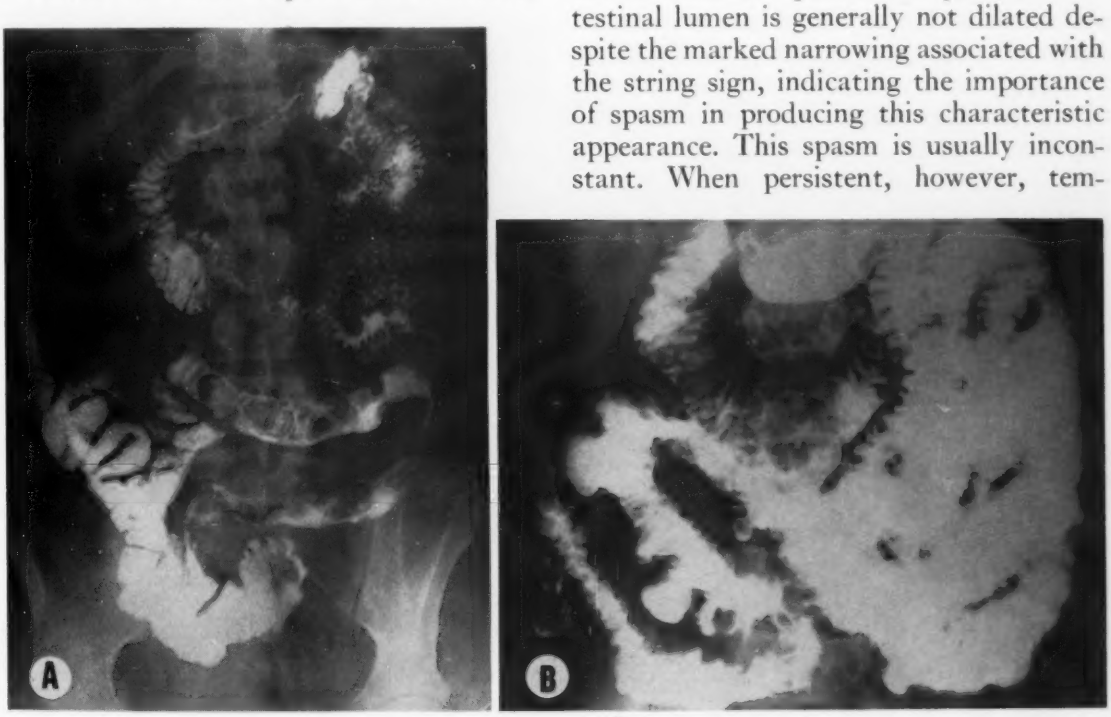


Fig. 4*A*. Unusually large inflammatory polyps in the distal jejunum and proximal ileum. Diagnosis: Nonstenotic type of regional enteritis.

Fig. 4*B*. Normal bowel protrudes between thickened blunted folds and suggests the presence of numerous pseudodiverticula. These are probably due to eccentric skip areas. The loops of bowel are straightened, slightly separated and moderately rigid. Diagnosis: Nonstenotic type of regional enteritis.

porary proximal dilatation may occur with symptoms of obstruction. In the stenotic phase there is constant proximal dilatation which may be accentuated by spasm secondary to ulceration. Despite the narrowing complete intestinal obstruction is rare. The recognition of the fact that the string sign does not always indicate marked fibrosis and stenosis is important as exclusion operations during the stage of marked ulceration and activity are often followed by a high degree of recurrence.

Because of the marked ulceration, fistulae and perforation (Fig. 8*A* and 8*B*), thickening of the mesentery and separation of the loops of bowel are more frequent in the distal ileum than in the remainder of the small bowel. A possible explanation of the marked ulceration in the distal ileum may be the higher bacterial content in this area.

Deformity of the cecum and ascending colon is not unusual with involvement of the terminal ileum. The deformity is most often due to the intense inflammatory process in the mesentery and to fistulae arising from the terminal ileum and extending into the right side of the colon (Fig. 7*B*). In this series of cases regional enteritis usually ended abruptly at the ileocecal valve.

Regional enteritis involving the terminal ileum does not always produce marked ulceration. There are many instances where the inflammatory process is minimal. In these patients the roentgen findings reveal slight rigidity, slight lack of distensibility and slight serration of the contour of the bowel.

Ileojejunitis. One hundred and two cases of ileojejunitis were studied. The distal half of jejunum and proximal half of ileum are characteristically involved (Fig. 5*A* and 5*B*). Skip areas are frequent. Large inflammatory polyps may be noted (Fig. 4*A*). Ulceration is not as marked as in the terminal ileum and fistula formation is less frequent. Because of the extent of involvement operative intervention until recently has been rare and long follow-up of the natural course of the disease has been possible. The transition from the nonstenotic to the stenotic stage could be observed and in the average case the interval

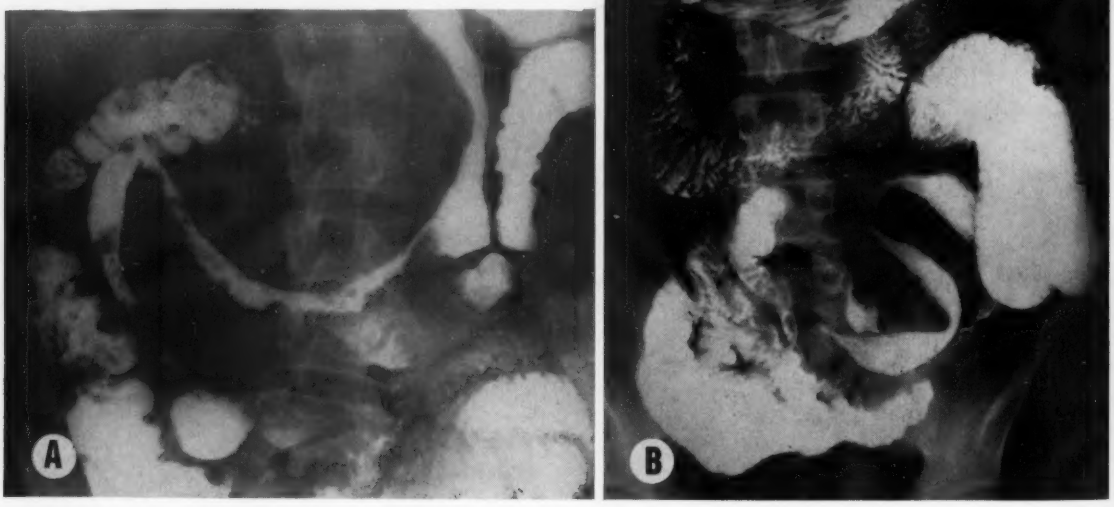


FIG. 5*A*. The mucosa is ulcerated and inflammatory polyps are present. The loop is rigid, narrowed and surrounds an inflammatory mass. There is slight proximal dilatation, Diagnosis: Stenotic type of regional enteritis.

FIG. 5*B*. Long, rigid, narrowed segments are noted in the distal jejunum and proximal ileum with proximal dilatation. Diagnosis: Stenotic type of regional enteritis.

was approximately four to five years.

In the last five years 9 patients with extensive involvement of the small bowel had massive resections of varying segments of jejunum and ileum. Four patients are well. Two patients have electrolyte and nutritional disturbances that are difficult to control. One patient developed ulcerative colitis and 2 patients developed recurrent lesions in the distal bowel.

It is of interest that many of these patients with such extensive involvement of the intestinal tract and despite numerous episodes of partial intestinal obstruction can maintain an adequate nutritional status. This group of cases is reported in detail in a previous communication.[12]

Jejunum. Twelve cases isolated to the jejunum were studied. In every patient there is marked stenosis. Three types of stenosis have been recognized: a single area of stenosis with proximal dilatation; a second form with two areas of narrowing and marked dilatation of the intestine between, simulating a closed loop obstruction; and a third type with multiple stenotic segments and intervening dilatation of the intestinal loops (Fig. 9*A*). The length of stenosis has been short varying from one to three inches. Ulceration is minimal. Fistula formation is uncommon and the thickening of the mesentery is not as marked as in the remainder of the small intestine. Sidetracking operations have been successful in every case but one, again pointing to the fact that recurrences are more frequent when the operation is performed in the presence of marked ulceration and fistula formation.

Isolated jejunitis in the nonstenotic stage has not been observed. These patients apparently have minimal symptoms for many years and are not investigated until partial obstruction supervenes.

Duodenum. Four cases were studied and the findings in the duodenum are essentially similar to those in the jejunum. (Fig.

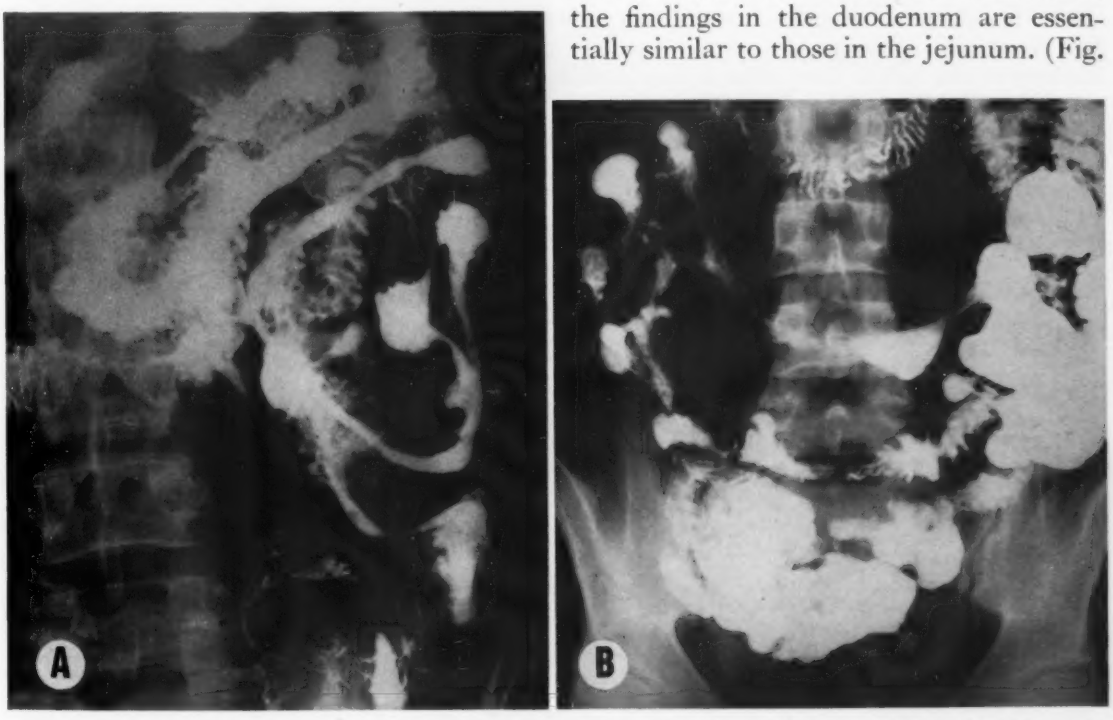


Fig. 6*A*. The entire small bowel is involved with alternating areas of constriction and dilatation. Diagnosis: Stenotic type of regional enteritis.

Fig. 6*B*. Long segments of constricted bowel alternate with small areas of dilatation. In the constricted segments the mucosa is ulcerated and presents a reticulated pattern. Diagnosis: Stenotic type of regional enteritis.

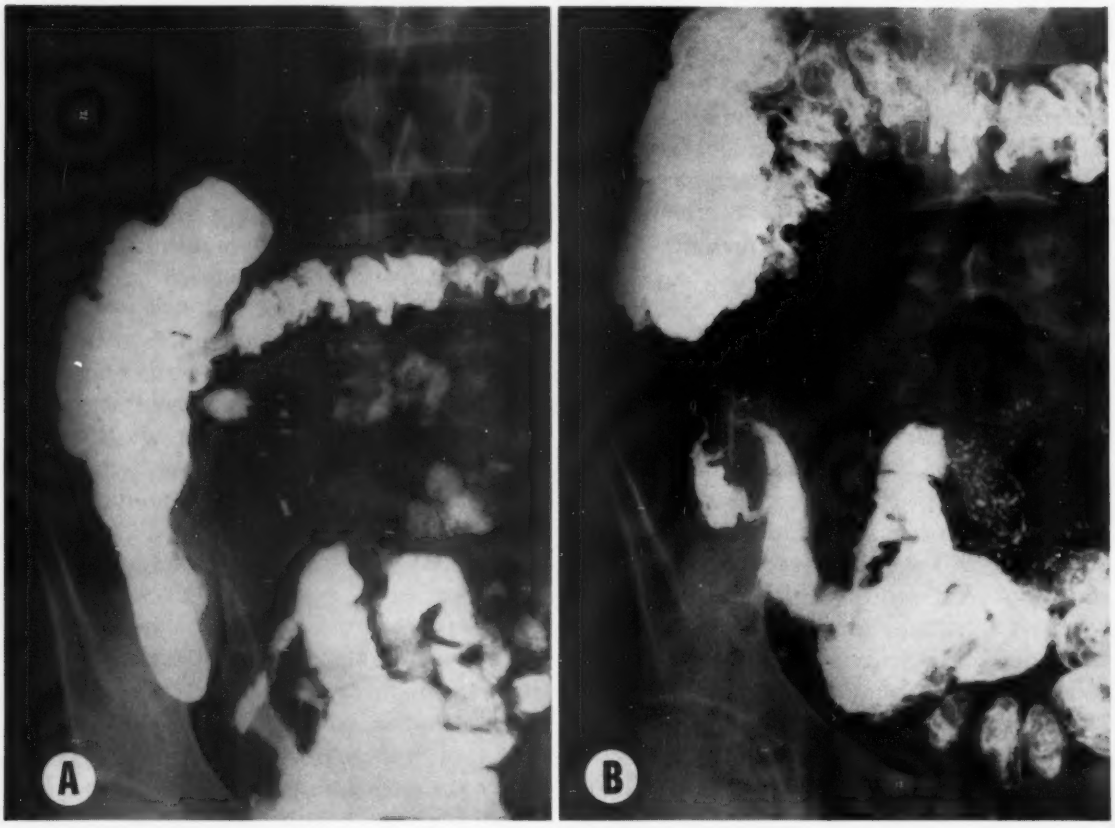


FIG. 7*A*. "String sign" in regional enteritis.

FIG. 7*B*. Same patient as 7*A* a few minutes later. There is more distensibility of the distal ileum. The cecum and ascending colon are more constricted. The findings in the ileum are due to marked ulceration and secondary spasm. The narrowing and rigidity of the cecum and ascending colon are due to a fistula from the distal portion of the terminal ileum (found at operation).

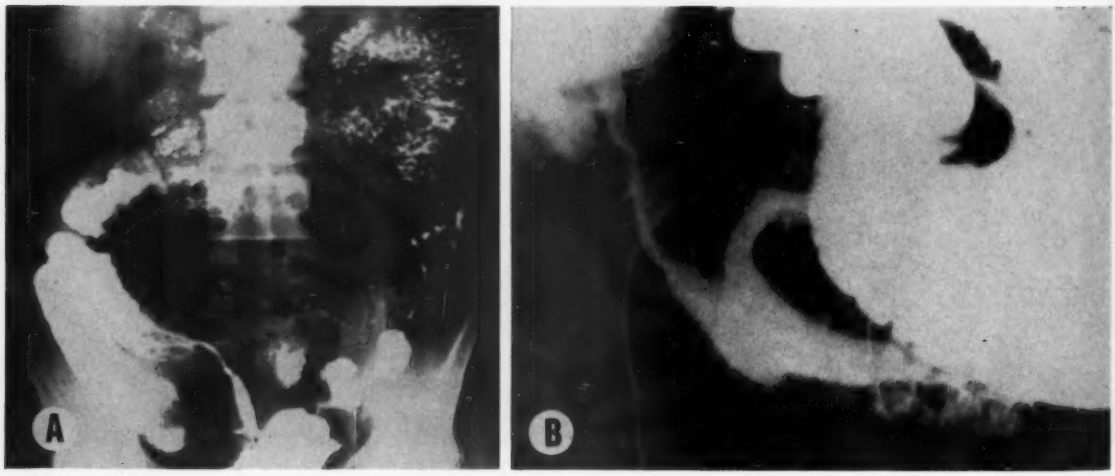


FIG. 8*A*. Barium passes from the involved terminal ileum into the sigmoid through a large fistulous communication. Diagnosis: Regional enteritis with a large fistulous tract extending into the sigmoid.

FIG. 8*B*. Numerous fistulae extend from the terminal ileum into the mesentery and toward the bladder. Diagnosis: Regional enteritis with numerous fistulae.

9*B*). Moderate stenosis is present and the ulcerated areas are seldom deep or marked. Two of these patients have an associated terminal ileitis.

Recurrent Ileitis. Following side-tracking operations for regional enteritis, recurrent disease in the new terminal ileum is not infrequent. Statistics as to the frequency of this recurrent lesion vary between 20 and 60 per cent. Recurrent disease is most frequent during the first year. However, many years may elapse before recurrence takes place. Recurrences also appear to be more frequent in those patients where the initial lesion reveals marked ulceration with fistulae formation.

In the cases where operation is performed because of stenosis, the recurrence rate appears to be less. The length of the lesion has no effect on the recurrence rate. In general, the recurrent lesion acts similarly to the original process. The colon is rarely invaded and the usual site is the new terminal ileum (Fig. 10*A* and 10*B*).

Healing in Regional Enteritis. While spontaneous remissions and clinical improvement following a variety of operative and conservative therapeutic procedures are frequently observed, there is not a great deal of information regarding roentgenologic and pathologic healing. Occasionally, patients who undergo re-exploration following ileocolostomy show a remarkable degree of healing of the involved segments of intestine and mesentery. The ulcerations may disappear and the fistulae heal. Demonstration of improvement without operation is rare in this series. Three cases, however, have been observed where remarkable roentgen changes indicating healing occurred (Fig. 11*A* and 11*B*). Since none of these patients were explored, it is possible that they represent instances of a different inflammatory process, although the roentgen features are those usually associated with regional enteritis. Despite these instances of improvement, the large majority of cases demonstrate the disease

Fig. 9*A*. Alternating segments of constriction and dilatation in the jejunum starting at the ligament of Treitz. There is dilatation of the duodenum. Diagnosis: Regional enteritis involving the jejunum with obstruction.

Fig. 9*B*. Duodenitis. Patient also has terminal ileitis.

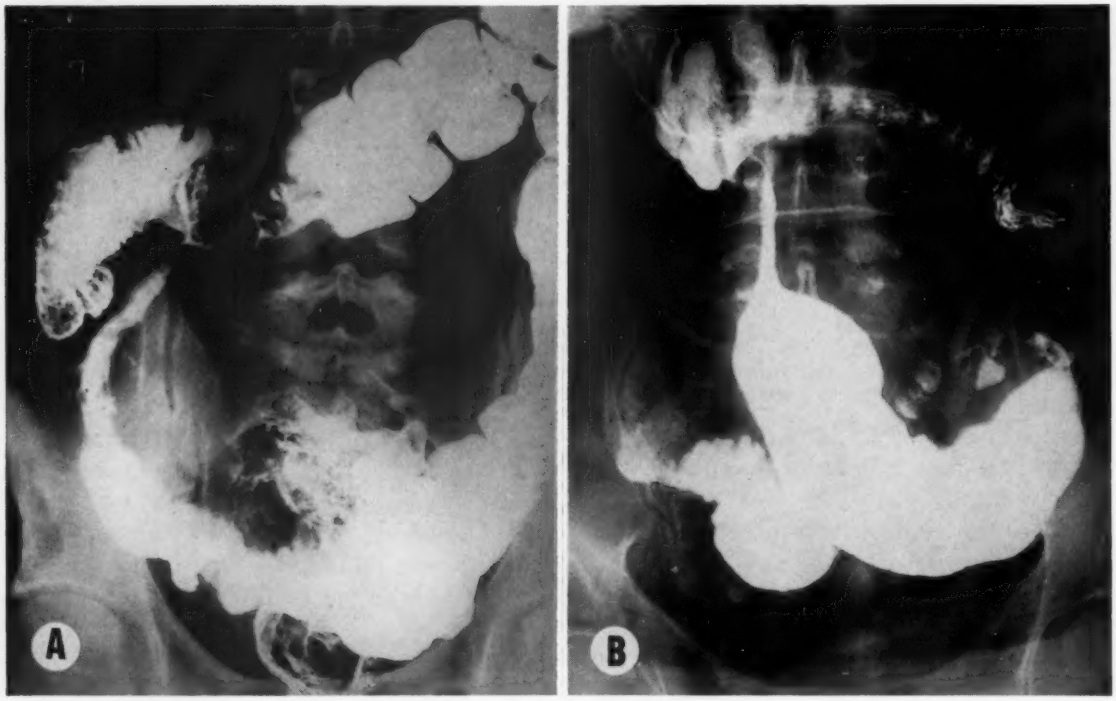


FIG. 10*A*. Status: Post-ileotransverse colostomy with recurrent ileitis involving the new terminal ileum, characterized by narrowing, rigidity, ulceration of the mucosa and small inflammatory polyps. The defect in the colon adjacent to the stoma was due to adhesions.

FIG. 10*B*. Status: Post-ileotransverse colostomy. There is marked narrowing of the ileum adjacent to the stoma for a distance of 6 cm. and considerable dilatation proximal to this narrowed segment. Other alternating areas of constriction and dilatation are seen proximal to this loop.

to be progressive. Many years, however, may elapse before stenosis supervenes. It should be pointed out that secondary changes may occur in patients who are clinically improved, notably stenosing lesions and permanent loss of the normal small intestinal structure. These changes are all due to healing with scar formation.

Combined forms of regional enteritis and diffuse ulcerative colitis occurring simultaneously are uncommon. However, twenty cases of combined regional enteritis and right sided (regional, segmental) ulcerative colitis in which the ileum, cecum and ascending colon are involved have been observed. The roentgen changes in the small intestine are characteristic of regional enteritis in every case. The alterations noted roentgenologically in the colon are largely those of nonspecific ulcerative colitis. In four cases, however, the changes differed from those usually seen and resembled closely the appearance of the affected ileum in that stenosis was a conspicuous feature. It is of interest that several patients who had an ileocolic resection for regional enteritis developed nonspecific ulcerative colitis in the remaining colon.

Nonspecific inflammatory disease of the ileum other than regional enteritis can occur. The frequency of involvement of the ileum in nonspecific ulcerative colitis varies from 8 to 20 per cent. The involvement of the ileum is usually minimal in extent and severity and has been considered as "backwash" of the colitis through the ileocecal valve. On occasion such ileitis may be severe and extend backward for varying lengths in continuity with the affected colon or may occasionally exhibit skip lesions. Fistulae are uncommon. Pathologically the lesion is similar to that in the colon in that the mucosa is

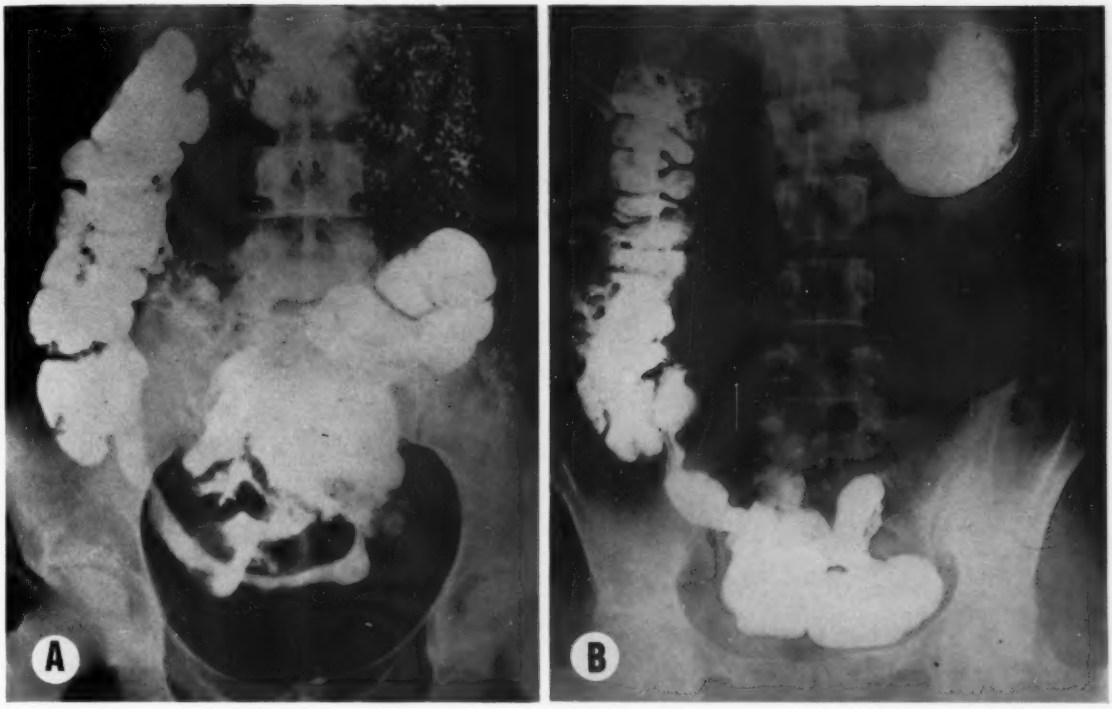


Fig. 11*A*. Regional enteritis involving the distal 18 inches of ileum.
Fig. 11*B*. Same patient as 11A. There is marked resolution in the appearance of the lesion following six months of therapy.

primarily involved. The roentgen appearance of the affected ileum is that of an incompletely distensible segment of bowel with varying degrees of ulceration of the mucosa. The rigidity and narrowing usually associated with regional enteritis are rarely observed.

Inflammatory disease of the ileum may be associated with right-sided (regional, segmental) ulcerative colitis. The roentgen appearance of the colon in this disease is the same as in nonspecific universal ulcerative colitis. The lesion in the ileum is continuous with the lesion in the colon. In general, the roentgen findings in the ileum simulate those in the colon but to a lesser degree. The length of affected ileum varies from 4 to 30 centimeters. In most of the cases the ileum is incompletely distensible and reveals alterations in mucosal pattern and contour consisting of thickening of the mucosal folds, slight irregularity of the bowel lumen and increased secretions (Fig. 12*A*). In the severer cases cobblestoning or a cast-like pattern due to further ulceration and denudation of the mucosa occurs. Occasionally spasm may be so severe that an appearance similar to the "string sign" is noted and therefore at times it is difficult to distinguish this process from regional enteritis. However, the lack of fistula formation, the lesser degree of narrowing of the affected segment with absence of dilatation of the proximal small bowel, the lack of evidence of thickening of the mesentery and the absence of a pressure defect on the medial wall of the cecum help to distinguish the changes in nonspecific ulcerative enteritis from those seen in regional enteritis. The term ileocolitis has frequently been used to describe this group of cases. There is considerable divergence of opinion regarding the interpretation of the pathologic alterations in this disease. Up to the present time we have considered this disease similar to nonspecific universal ulcerative colitis, confined to the right side of the colon with extension into the ileum.

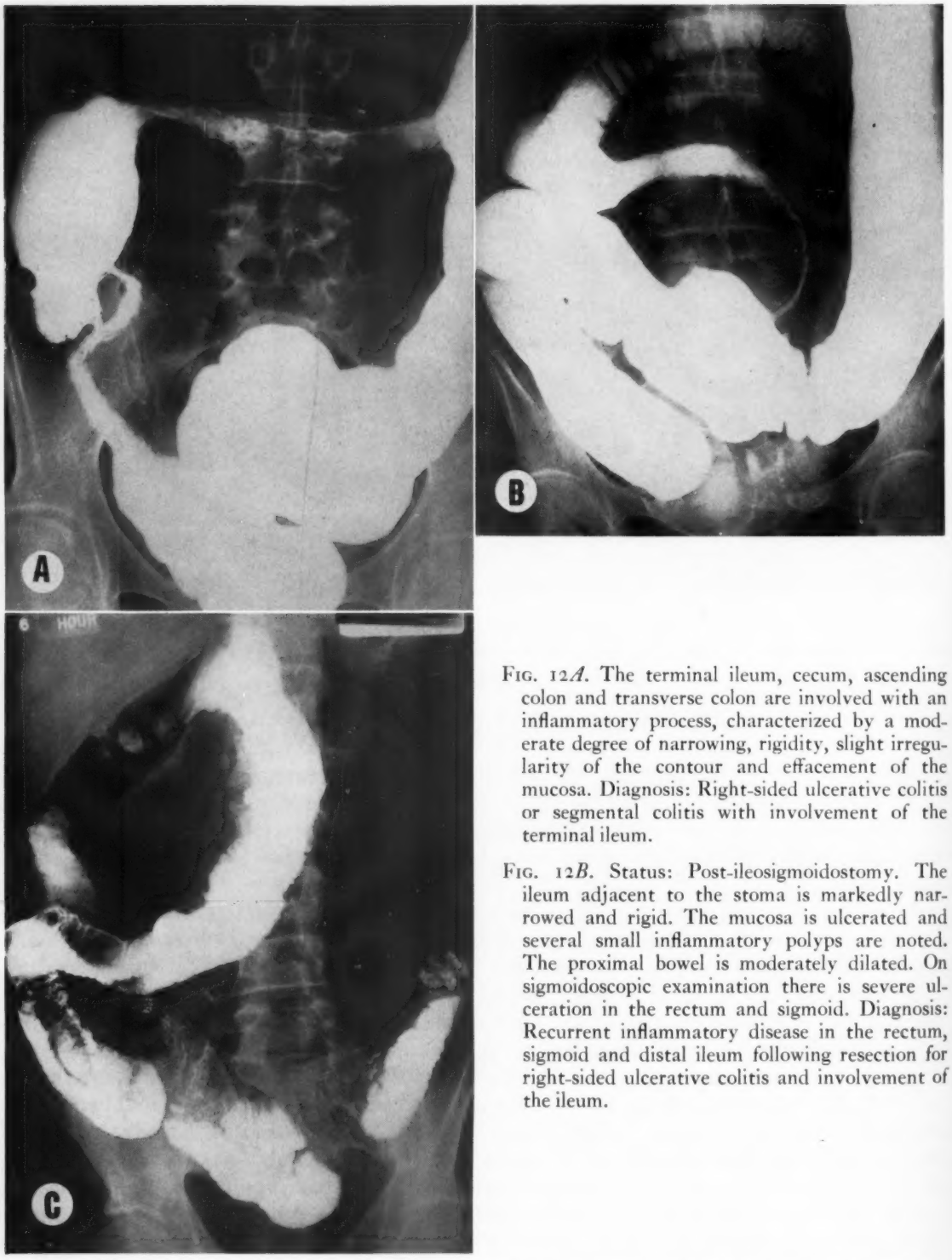


FIG. 12*A*. The terminal ileum, cecum, ascending colon and transverse colon are involved with an inflammatory process, characterized by a moderate degree of narrowing, rigidity, slight irregularity of the contour and effacement of the mucosa. Diagnosis: Right-sided ulcerative colitis or segmental colitis with involvement of the terminal ileum.

FIG. 12*B*. Status: Post-ileosigmoidostomy. The ileum adjacent to the stoma is markedly narrowed and rigid. The mucosa is ulcerated and several small inflammatory polyps are noted. The proximal bowel is moderately dilated. On sigmoidoscopic examination there is severe ulceration in the rectum and sigmoid. Diagnosis: Recurrent inflammatory disease in the rectum, sigmoid and distal ileum following resection for right-sided ulcerative colitis and involvement of the ileum.

FIG. 12*C*. Status: Post-terminal ileostomy. There is moderate dilatation of the distal loop of ileum with marked irritability, thickening and blunting of the mucosal folds and irregularity of the contour. Diagnosis: Nonspecific inflammatory disease of the ileum following ileostomy for right-sided ulcerative colitis with extension into the ileum.

Whether or not the disease in the ileum is due to "backwash" or an intrinsic part of the disease process has not been determined as in many cases the ileal alterations are extensive and well established when the patient is first observed. The importance of demonstrating the extent of the ileal involvement should be stressed in those patients in whom surgical procedures are contemplated as it is likely that some of the cases of recurrent disease may be due to unrecognized disease left in the abdomen.

Inflammatory disease of the ileum may occur after ileocolostomy for right-sided ulcerative colitis. Recurrences have been noted despite the fact that biopsy of the anastomosed loop of ileum at the time of operation reveals no evidence of inflammatory disease. Usually they have occurred first in the colon followed by extension into the adjacent ileum. The roentgen appearance of recurrent disease in the ileum is similar to the appearance noted preoperatively (Fig. 12*B*).

Inflammatory disease of the ileum can also occur after terminal ileostomy for ulcerative colitis, even with a normal terminal ileal segment demonstrated microscopically at the time of operation. In this group of cases, stomal difficulty with partial or complete intestinal obstruction is frequently encountered. The roentgen findings in most cases consist of thickening and blunting of the mucosal folds, some rigidity of the bowel wall and dilatation of the lumen. Excessive secretions are usually present. Resection of the affected ileum in these cases reveals a nonspecific inflammatory process of the mucosa with ulceration and some fibrosis of the remaining coats of the intestinal wall.

The pathologic changes usually associated with regional enteritis are not present. The alterations observed may be secondary to obstruction at the stoma; however, the possibility that intrinsic non-specific inflammatory disease in the bowel wall may be the primary reason for the obstruction cannot be excluded (Fig. 12*C*).

SUMMARY

1. The most common nonspecific inflammatory lesion of the small intestine is regional enteritis.

2. Regional enteritis may affect any portion of the small intestine. However, it most commonly involves the terminal ileum. Since stenosis is such a prominent feature of this disease the roentgen findings have been conveniently divided into the nonstenotic and stenotic stages. The roentgen changes from the earliest alterations to the late stenotic stage as well as the changes characteristic of the site of involvement are described.

3. The length of involvement is usually determined on the initial examination. Proximal and distal extension without operation is uncommon.

4. The large majority of cases demonstrate the disease to be progressive. However, many years may elapse before stenosis supervenes. Resolution in regional enteritis is infrequent.

5. Regional enteritis extending into the colon is uncommon in our experience.

6. Recurrent ileitis following side-tracking procedures with and without resection of the diseased bowel occurs with disturbing frequency. Again, the new terminal ileum is involved without involvement of the colon.

7. The combination of regional enteritis and right-sided ulcerative colitis was seen twenty times in this series of cases. Diffuse ulcerative colitis with regional enteritis was uncommon without previous operative intervention.

8. Nonspecific ulcerative colitis may involve the distal portion of ileum, and the small intestinal lesion must then be differentiated from regional enteritis.

9. Specific inflammations of the small intestine which can be recognized on the roentgen examination are uncommon in this area.

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INTESTINAL OBSTRUCTION*

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THE roentgen ray examination is useful in establishing the existence, location, degree and complications of intestinal obstruction and in observing progress under treatment, but this is true only when it is used in conjunction with the history and the physical examination. Employed alone it may be dangerously misleading. Of course, roentgenologic diagnosis is simply a part of the "inspection" which together with palpation and auscultation constitutes the physical examination, but in many situations it is so incisive as to dwarf the importance of the other two members of the triad. In the case of the skeleton and the lungs, roentgenology unaided by other procedures usually can be depended on to show whether lesions are present or not and frequently to establish the etiology, but the situation is quite otherwise in intestinal obstruction. Here the structures under inspection have little inherent density difference and usually artificial contrast media are not employed except in the colon, where fluoroscopy and filming during a carefully administered barium enema are superior to plain filming or filming after oral barium.

The roentgen criteria of intestinal obstruction with and without strangulation, peritoneal effusion or pneumoperitoneum have been described by many writers, including Case,[1] Kloiber,[5] Rigler,[6,8] Mondor et al.,[7] and Frimann-Dahl,[4] and a monograph by Wangensteen[9] deals exhaustively with the clinical aspect of these conditions. The purpose of the present paper is to recapitulate the clinical and roentgenologic criteria of intestinal obstruction, to emphasize the shortcomings of roentgenology as a means of detecting arterial or venous thrombosis of mesenteric vessels, and to report experimental work on air injected into the peritoneal cavity.

Anatomically the two major divisions of the small intestine pass insensibly from one into the other without noticeable change to indicate the point of transition, but by convention the proximal two-fifths is known as jejunum, the distal three-fifths as ileum, and upper jejunum differs significantly from lower ileum. A typical segment of jejunum has a slightly greater caliber and thicker wall than ileum and its plicae circulares (synonyms: valvulae conniventes, Kerkring's valves, Kerkring's folds) are better developed, while typical ileum is narrower and thinner than jejunum, has fewer and smaller plicae even in its proximal half and usually none in its distal half.[2] The root of the mesentery of the small intestine, approximately 7 inches in length, is attached to the posterior abdominal wall along a line that starts above at the left side of the body of the second lumbar vertebra and passes obliquely downward and to the right to terminate in the right iliac fossa.

Chyme intimately mixed with small bubbles of air on leaving the duodenum passes rapidly through the upper jejunum and then somewhat more slowly through the ileum where it is converted to feces. Large amounts of digestive juice are added along the way but this is balanced by the absorption of water and chyle and normally there is no distention of the bowel. The capacity of the jejunum and ileum is so great that in the absence of obstruction those organs do not become distended even when experimental subjects swallow large amounts of air (Fig. 1) or fluid or when patients with subtotal gastric resection ingest rapidly several liters of barium sulfate mixture. When, however, the forward movement of intestinal content is halted

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FIG. 1. Ingested Air Filling But Not Distending Small Bowel. F.T., M-31, No. 604754. This normal volunteer, whose bowel was almost free of air at the start of the experiment, ingested 2,000 cc. of water during a period of ten minutes and then swallowed as much air as possible. Films were made at frequent intervals and all of them, like this one made two and one half hours after the beginning of the experiment, show air throughout the small bowel but no distention of the sort encountered in intestinal obstruction. Similar collections of air are common in bedridden patients coming to the radiologic department for examination of the urinary tract, spine, etc.

and air and fluid continue to be introduced from above the bowel proximal to the obstruction gradually becomes distended and the small air bubbles separate out from the fluid and coalesce. Whether the obstructing agent be a band of adhesions, volvulus, intussusception, a large gallstone, a mass of ascaris worms or an unreduced hernia, the patient usually will suffer pain, vomiting and distention; but, of course, these same complaints are encountered in many other acute abdominal conditions, including postoperative adynamic ileus. Obstruction in itself is fairly well tolerated until intestinal distention becomes pronounced or until the blood supply to a segment of bowel is interrupted—a condition called strangulation. In volvulus, intussusception and unreduced hernia, the mechanical situation that causes the blockage of the lumen may obstruct the mesenteric vessels as well, but distention alone is able to block them if it is of severe grade and long duration. Even in the absence of mechanical obstruction there may be blockage of mensenteric arteries by embolism in heart disease or of the veins by thrombosis secondary to regional infection or incidental to thrombophlebitis elsewhere in the body. When a segment of small bowel is obstructed at two points, the condition is known as "closed-loop" obstruction. The bowel distal to such a loop usually empties normally and that proximal to it usually is distended except during those intervals when vomiting or continuous suction has removed the dammed up air and fluid.

Wangensteen emphasizes the importance of intestinal colic as an indicator of the presence or absence of mechanical obstruction. The examiner listens to the abdomen with a stethoscope and diagnoses intestinal colic when borborygmia coincide with the height of attacks of cramping pain. In the absence of fever, diarrhea or a history of food allergy or dietary indiscretion, intestinal colic suggests a diagnosis of mechanical obstruction and this diagnosis is strongly confirmed if there is frequent copious vomiting of yellowish brown material. Usually when obstruction is complicated by strangulation, internal hemorrhage occurs eventually with the usual systemic sequelae of blood loss plus evidence of free fluid in the peritoneal cavity and pain that persists in the intervals between seizures of colic.

Roentgenograms of the abdomen made without contrast media provide meager and frequently misleading evidence as to the location and condition of the normal jejunum and ileum, but when those organs are distended with gas, their roentgen visibility increases and except for direct inspection through an abdominal incision the roentgenogram is the most dependable means of establishing the presence of in-

testinal distention. Films made with the patient recumbent possess advantages over all others and should never be omitted, regardless of what other special films may be made. Of course, one should use a grid, either moving or stationary, and a roentgen ray intensity great enough to allow brief exposures. Stereoscopy sometimes is helpful and would be more important if manufacturers would produce apparatus for the rapid shifting of films and tube, such as is regularly available for chest filming. In the recumbent position gravity flattens the abdomen, and when in addition a compression band is applied there is a significant decrease in the thickness of the tissue to be penetrated, with corresponding reduction in scatter. Any gas-filled loops of intestine that are free to move will float to the anterior abdominal wall and any free peritoneal fluid that may be present will sink to the flanks. The prone position places bowel closer to the film, it is true, but tends to distort it by pressure and almost always is uncomfortable for the patient, the discomfort being bad in itself and also increasing the likelihood of movement during exposure. Occasionally the amount of air in fluid-distended loops is so small that it may be overlooked unless special films are made with the axis of the cone of radiation directed tangentially to the interface between air and fluid. This may be accomplished by having the patient stand facing the film, lie on his back with the film at his side, or lie on his side with the film against the anterior abdominal wall—in each instance with the axis of radiation

Fig. 2. Small Bowel Distention Caused by Obstruction. H.H., F-78, No. 581627. A loop of mid-ileum, incarcerated and strangulated in the sac of a femoral hernia, has obstructed the small bowel. The distended jejunum is arranged in transverse layers in the mid-abdomen forming the so-called "ladder rungs" and the stretched-out plicae circulares produce the so-called "herring-bone" pattern. The colon and the ileum distal to the obstruction are not distended.

Fig. 3. Closed-Loop Obstruction of Ileum with Fluid-Filled Loop. L.R.S., F-29, No. 427382. A band of adhesions near the cecum has constricted at two points a 30 cm. segment of ileum. The obstructed loop is full of fluid and the bowel proximal to it is distended with air. In this supine film, made a few hours before operation, the fluid-filled segment of ileum shows as a pseudotumor in the lower left quadrant with gas-filled loops of small bowel above.

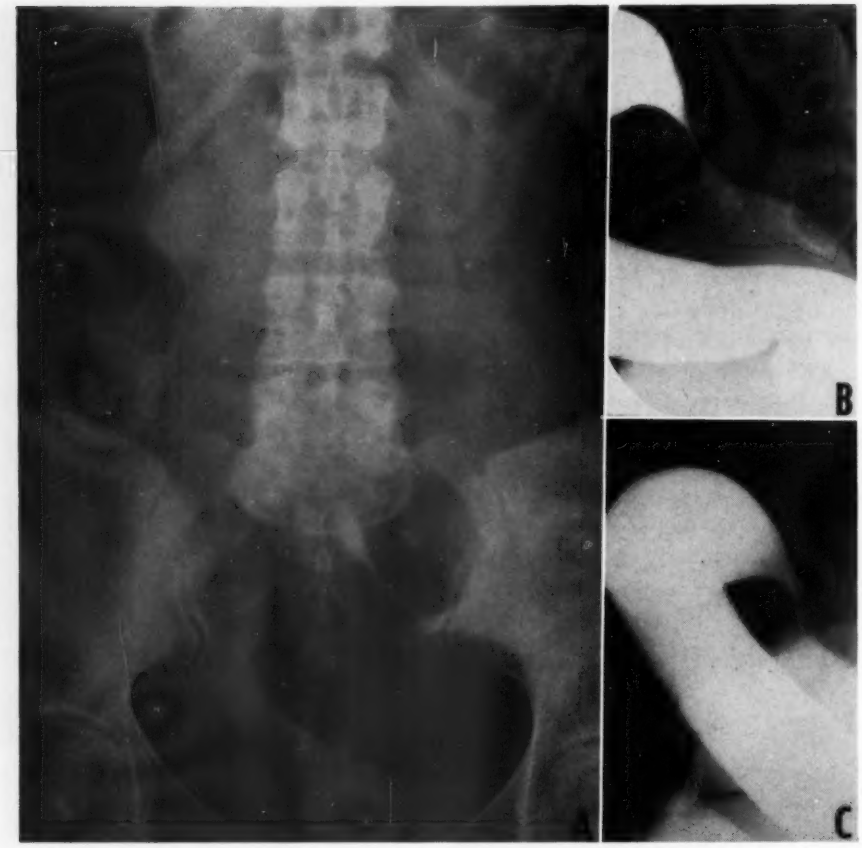

FIG. 4. Volvulus of Sigmoid Causing Closed-Loop Obstruction with Gaseous Distention of the Loop. E.H., F-55, No. 553207. (*A*) One of a pair of stereoscopic films made twenty-four hours after onset of vomiting and colicky pain and three hours before administration of barium enema. Volvulus of redundant loop of sigmoid has caused a closed-loop obstruction and air trapped in the loop can be seen at the left side of the pelvis. (*B* and *C*) "Spot" films made three hours later during administration of barium enema show typical volvulus of the sigmoid. At subsequent surgical exploration sigmoid blood supply was intact but pressure from the distended loop of colon was beginning to embarrass small bowel vessels.

parallel with the floor and perpendicular to the film. In such films the interfaces between air above and fluid below constitute the "fluid levels" about which so much has been written. In a typical case of mechanical obstruction low in the small intestine, films made with the patient in the recumbent position will show gas-distended loops of jejunum and ileum arranged in transverse layers across the central portion of the abdomen with little gas in the peripherally lying colon. Loops of jejunum will be characterized by the stretched-out plicae circulares, the so-called "herring bone" pattern, and by their tendency to locate toward the upper left of the central portion of the abdomen (Fig. 2). Ileal loops will be lower and farther to the right and will show fewer plicae circulares. When jejunal distention is maximum the plicae may be mistaken for the haustra of distended colon, but usually the two organs can be differentiated. In closed-loop obstruction with no air in the distended segment the lesion sometimes can be detected in films by its resemblance to a neoplasm displacing other abdominal viscera (Fig. 3) but the roentgen diagnosis is easier when the closed loop contains air (Fig. 4). Rigler has pointed out that when an air-filled closed loop is folded on itself one sometimes sees two oval gas shadows

separated by a dense band (representing two adjacent walls of bowel) which may be likened to a gigantic coffee bean.

In the colon the common obstructing agent is neoplasm, with volvulus of the sigmoid or cecum a much less frequent cause. Except for those cases in which the obstruction is high in the cecum, abdominal distention becomes severe; but usually the ileum is able to continue to discharge its contents into the cecum, and the ileocecal valve (Fig. 5) prevents reflux of fluid and gas into the small bowel. As a result nausea, vomiting and small bowel distention are minimal or absent (Fig. 6). Radiologists who regularly attempt to fill the terminal ileum refluxly during the course of a barium enema and usually succeed in doing so are apt to be skeptical as to the ability of the ileocecal value to prevent reflux from the colon but, as Wangensteen points out, the hydrostatic pressure employed during a barium enema is several times greater than the maximum pressure encountered in colon distention. In some patients the valve is incompetent because of congenital shortness of the lower lip and in such cases an obstructing lesion of the colon will distend not only that portion of the colon lying proximal to the obstruction but the jejunum and ileum as well (Fig. 7).

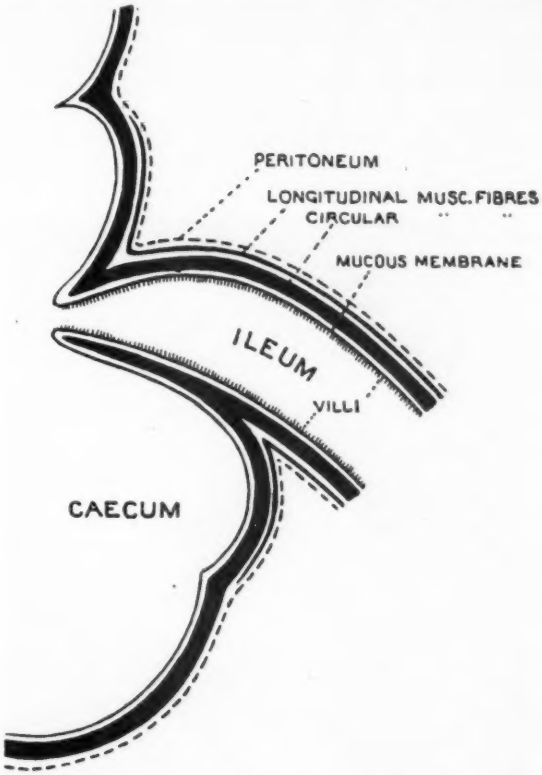

FIG. 5. Ileocecal Valve.[9] In the normal subject the oblique invagination of the ileum into the cecum together with the long inferior lip of the ileum makes for an efficient valve. When there is congenital shortness of this lip the valve may be incompetent.

FIG. 6. Competent Ileocecal Valve. T.S., M-51, No. 408994. Recurrent carcinoma obstructs the sigmoid causing gross gaseous distention of the entire colon, but in spite of this distention the ileum is able to continue to empty into the cecum and a competent ileocecal valve prevents reflux of air and fluid.

It would be gratifying if roentgenology were able to do as well in diagnosing the existence of strangulation as it does in demonstrating intestinal distention, but unfortunately such is not the case. To establish the existence of volvulus or of closed-loop obstruction is to suggest strongly the probability of strangulation and that probability is increased when it

FIG. 7. Incompetent Ileocecal Valve. L.A., F-72, No. 31261. In this case of obstruction of the sigmoid by uterine carcinoma, there is distention not only of the colon but also of the small bowel. Autopsy demonstrated the absence of obstructing lesions in jejunum or ileum, but unfortunately the ileocecal valve was not examined for evidence of congenital shortness of the inferior lip. It is assumed that the valve was incompetent and that this explains the distention of the small bowel along with the colon.

FIG. 8. Venous Mesenteric Thrombosis of Ileum Not Producing Obstruction. R.F., F-54, No. 528487. There is venous thrombosis and gangrene of three feet of terminal ileum (proved at operation five days after this film was made) and yet barium administered two days earlier for examination of the stomach has passed through the small bowel and colon without delay.

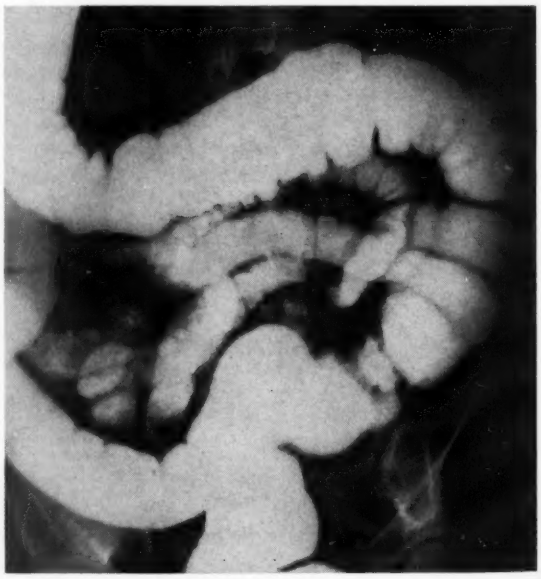

FIG. 9. Venous Mesenteric Thrombosis of Ileum with Normal Appearing Mucosa. (Same Case as Figure 8.) The gangrenous terminal ileum appears normal when it is filled refluxly during examination of the colon. It emptied normally when the enema was expelled.

can be shown that such loops are fixed in position and tender to palpation. Strangulation, however, is not invariably present in volvulus and closed-loop obstruction and sometimes the occlusion of mesenteric vessels does not produce pain, tenderness or fixation. Particularly baffling for the radiologist are those cases of mesenteric thrombosis which are not the result of mechanical obstruction. Contrary to much of the older teaching, it is now recognized that such "dead" segments of small bowel may interfere surprisingly little with the forward movement of intestinal content (Fig. 8) and that their mucosa may look completely normal when the segments are filled refluxly during the administration of a barium enema (Fig. 9).

The clinical signs of perforation of a

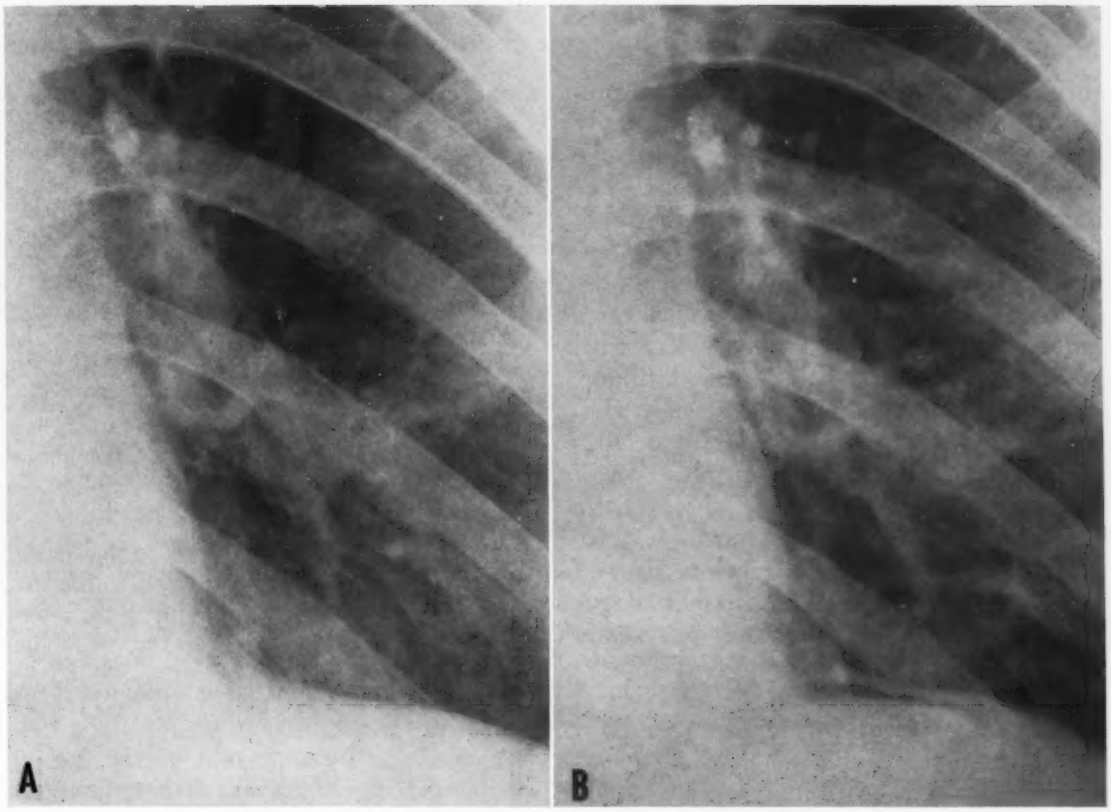

Fig. 10. Experimental Small Pneumoperitoneum Demonstrated in Erect Chest Film. R.M., M-36, No. 522857. (*A*) Before injection. No air is seen between liver and right leaf of diaphragm. (*B*) Twenty minutes following the injection of 2 cc. of air at McBurney's point. Air can be seen in the right subphrenic space.

viscus are so similar to those of acute intestinal obstruction that usually it is wise to look for evidence of both conditions whenever either is suspected. The roentgenologic evidence for perforation is, of course, the demonstration of pneumoperitoneum; and, unlike the case of air in distended bowel, free air in the peritoneal cavity will not show in recumbent films unless it is present in large amounts. One of the authors* recently has experimented with air injected into the peritoneal cavity at McBurney's point (Fig. 10). When the amount was 1 cc., gravitation of air from injection site to the subphrenic space occurred slowly so that it was necessary to have the subject assume the erect position for about thirty minutes before films became diagnostic but with 3 cc. injections gravitation was prompt. The nitrogen fraction of the peritoneal air was absorbed so slowly that for injections as small as 2 cc. a gas shadow could still be seen forty-two hours later.

When one is seeking evidence of pneumoperitoneum incident to a perforated viscus, its is desirable to make stereoscopic chest films with the patient standing facing the film and to have him sit or stand for half an hours before the filming is done but, of course, many patients will be so ill that they can barely stand for the filming itself. For patients too ill to stand at all or even to sit, free peritoneal air sometimes will show in special horizontal films in which the axis of the cone of radiation is directed parallel with the floor and perpendicular to the film.

* R. M. served as the subject, Dr. S. W. Nelson performed the injections.

CONCLUSIONS

1. The roentgenogram is the best non-surgical means of demonstrating the presence of small bowel distention, but usually non-roentgenologic procedures must be employed to differentiate the distention of obstruction from that of several other conditions, including adynamic ileus.

2. "Closed-loop" obstruction is most easily diagnosed when the loop contains air, but even when it is filled solely with fluid, the loop may show as a pseudotumor.

3. Occlusion of mesenteric vessels, whether arterial or venous and whether occurring independently or as a complication of obstruction, unfortunately gives little roentgen evidence of its presence.

4. In obstruction of the colon, usually the ileocecal valve prevents reflux of gas and fluid into the small bowel but sometimes the valve is congenitally incompetent.

5. Pneumoperitoneum is a frequent but by no means invariable concomitant of intestinal perforation and properly made chest films can be depended upon to demonstrate even very small amounts of free peritoneal air.

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DISCUSSION OF SYMPOSIUM ON INFLAMMATORY LESIONS OF THE GASTROINTESTINAL TRACT

(Papers by Drs. French and Vander, Drs. Fred Hodges and Rubin, Drs. Marshak and Wolf, and Drs. Paul Hodges and Miller.)

DR. ROSS GOLDEN, Los Angeles, California. Any one of the papers in this symposium is well worthy of the undivided attention of a discusser.

I was much interested in Dr. French's demonstration of a barium granuloma. I have never seen one. I would like to ask Dr. French whether barium can get into the intestinal wall through intact normal mucosa or whether inflammation or some other injury must be present for this to occur.

Dr. Fred Hodges' discussion of esophagitis was very interesting. I would like to point out that regurgitation from the stomach into the esophagus can occur in the absence of a hiatus hernia. This regurgitation is most likely to occur when the patient turns over from a supine to a prone position, as I always have them do. If one looks quickly, as soon as this change in position is completed, one may see a bolus of barium in the esophagus, which is promptly expelled. Unless we look quickly, this will very likely not be noticed. I believe that I can show small hiatus hernias much better when the patient is in the prone position, with the weight of the body on the abdomen, and while he is in this position, have him swallow some barium. I have found that thick barium, that is, barium stirred up with water so thick that it will not fall off a spoon, is a very useful opaque medium for the examination of the esophageal mucosa.

Dr. Marshak has had probably the greatest experience with regional enteritis of anybody I know. He stated that irregularity of the mucosal folds is an early sign of regional en-

teritis. I would like to ask him just what he means by irregularity of these folds. Does he mean that single folds may show a variability in width, as one traces the shadow across the intestine, or does he mean that one fold may be different from another in width, so that one may be quite wide and another normal in width?

Dr. Marshak also commented on the ileitis which follows permanent ileostomy. This inflammation, as he pointed out, is quite different from that of regional enteritis, in that the inflammation is not associated with narrowing of the lumen. On the contrary, it is associated with dilatation of the intestine. This is quite a serious complication because of the electrolyte imbalance, which in some cases is the cause of death. I doubt that the trouble is caused by an obstruction at the stoma because in other conditions uncomplicated obstruction does not cause inflammation proximal to it. It seems to me more likely that the obstruction, which is due to spasm at the stoma, and the inflammation are manifestations of the same disorder.

Dr. Marshak also spoke about the differential diagnosis of regional enteritis and of sprue, or sprue-like syndromes. I have seen at least two cases of proved regional enteritis, which is what the pathologist called them, which followed nutritional disorders, manifested by the usual symptoms; namely, an abnormal small intestinal pattern and a flat blood sugar curve. Hence, it appears that one disease can develop into the other. In the study of organic diseases of the intestine by roentgen-ray method, I am now convinced that it is highly important to use one of the nonflocculating barium preparations.

Dr. Marshak pointed out the wide separation of some of the abnormal loops in regional enteritis. However, separation of loops of small intestine can occur without organic disease. Spasm involves the longitudinal as well as the circular muscle. We can easily see evidence of spasm of the circular muscle because the lumen is transversely narrowed. We cannot see evidence of spasm of the longitudinal muscle, but must assume that when tonus is abnormally high in one coat, the same condition must be present in the other coat. I have seen cases without evidence of inflammation in which there was marked hypertonic narrowing of the greater part of the small intestine, and separation of the loops occurred similar to that Dr. Marshak showed in regional enteritis. This separation must be due, in part, at least, to the contraction of the longitudinal muscle and thus to shortening of the intestine.

Intestinal obstruction is a big problem. Dr. Paul Hodges used the term "obstruction" as synonymous with what some of us have come to call mechanical ileus. Actually, ileus and obstruction are not synonymous; they are two different things. Obstruction, meaning a physical handicap to the passage of intestinal contents, can occur without ileus, for example, in the form of a kink or knuckle of intestine, due to peritoneal adhesions. This is sometimes called chronic obstruction. It may exist for a long time, even for years, without causing trouble because the intestine proximal to a mechanical handicap can compensate for it. Then for some unknown reason, this compensation fails, the intestine begins to dilate, and we have the roentgen-ray picture and the symptoms of mechanical ileus. Furthermore, a gradually increasing constriction such as an annular carcinoma can narrow the lumen down to 2 or 3 mm. without causing symptoms. The intestine compensates for this constriction as is shown by the hypertrophy of the muscle wall proximal to it. Then something happens so that the compensation breaks, and typical picture of ileus develops. It is of interest that, during the period before symptoms occur, the dilatation proximal to this kind of constriction may assume the proportions of that seen in typical ileus.

The experiments on injection of air into the abdominal cavity reported by Dr. Paul Hodges and Dr. Miller are very interesting. I had not realized that only 2 cc. of air in the peritoneal cavity could be seen.

It is obvious that these papers will be a permanent and valuable source of information about these interesting topics.

Dr. Leo G. Rigler, Minneapolis, Minnesota. Doctors Paul Hodges and Miller have presented a very comprehensive review of the usefulness of roentgen examination in some acute abdominal conditions. There are a few additional points which might be of value.

In occasional cases, a film made with the patient in the Trendelenburg position is worth while. Such films are especially valuable in those cases in which loops of bowel lie in the pelvis simulating a pelvic mass because of their

fluid content. The elevation of the pelvis may demonstrate fluid levels within areas which were formerly thought to be completely dense mass. In this way the distinction between pelvic inflammatory disease or a large pelvic mass and obstructed loops of bowel filled with fluid can some times be made.

I would emphasize the authors' point that in those difficult cases where there is a problem of differentiation between adynamic ileus and a mechanical obstruction of the large bowel a barium enema given with great caution under fluoroscopic control may be of considerable value. It is not at all rare to find the large bowel greatly distended with little or no distention of the small bowel suggesting strongly a mechanical obstruction low in the large bowel but, which on barium enema examination reveals no obstruction whatever, the distention being entirely the result of a neuromuscular dysfunction.

I agree completely with the authors' statement that "roentgen examination of the abdomen for the acute process must be combined intimately with the clinical history and physical findings in order to arrive at the most rational conclusions." Nevertheless, there are a number of conditions, one of which the authors have touched upon briefly, in which the roentgen examination is so characteristic as to indicate a specific diagnosis regardless of the clinical findings. The first condition the authors have touched upon, namely, strangulation obstruction. It should be noted that frequently in strangulation obstruction the evidences of obstruction itself are not entirely apparent and may be overlooked, but the demonstration by roentgen examination, either of the "coffee bean" sign, which I have described, or of the "pseudotumor" sign, which Frimann-Dahl has described, or the presence of fixed loops of small bowel without corresponding distention of other loops is in itself so highly indicative of the nature of the process as to warrant surgical intervention on the basis of the roentgenologic findings alone. The second condition is acute ileocecocolic intussusception, in which, when there is any doubt, the diagnosis can be readily established. Intussusception may exhibit a cone-shaped area of obstruction in a loop of gas-filled intestine near the right lower quadrant which is characteristic. Also, with the barium enema, it is rare that one fails to make the diagnosis correctly because of the characteristic picture of the intussuscipiens within the barium-filled colon. The third, extremely difficult type of intestinal obstruction to diagnose clinically is encountered in gallstone obstruction. Here the presence of linear or other areas of gas in the biliary tract, combined with the usual evidences of either chronic, intermittent, or acute intestinal obstruction should lead to the correct diagnosis, even though the stone itself is not demonstrable in the roentgenogram. It is notable that in all three of these situations, in which roentgen examination is so definitive, the indications for immediate surgical intervention are pressing.

The authors have pointed out the difficulties of roentgen diagnosis in vascular thrombosis. This has been our experience as well, and while there are some signs which are suggestive, in general, we believe that the absence of roentgenologic findings in the presence of an acute abdominal crisis should always suggest the diagnosis of mesenteric thrombosis.

In connection with the matter of perforation, it should be re-emphasized that a substantial percentage of perforations of the stomach and duodenum, probably 20 per cent, do not give evidences of visible gas under the diaphragms even though the patient has been put in the upright position for an appreciable period of time. There are many reasons for this. One, and perhaps the most important of all, is the tendency for such perforations to seal themselves off, after the escape of a small amount of fluid, with little or no gas. As a result, chemical and even bacterial peritonitis may supervene without the gas appearing under the diaphragms. It is not always possible to demonstrate the sealing of the perforation at surgical examination, but in many cases this has been clearly visible. There are other reasons, some of which depend upon the location of the perforation and also upon the presence or absence of diaphragmatic adhesions. We have found the lateral decubitus position, to which the authors refer briefly, of very great value in some cases; we have observed gas under the lateral abdominal wall in that position when we could not demonstrate it in the upright. I would repeat what I have said on previous occasions, namely, that it is possible to demonstrate pneumoperitoneum in the supine position, especially if gas is present in large amounts, by reason of the

double contrast which is afforded by the air outside the loop of bowel and inside of it, making the thickness of the bowel wall itself visible.

There are, of course, many other comments one could make. This is a very large subject and the authors have been able to touch upon each of its facets very briefly. It is gratifying to see how extensively roentgen examination without opaque medium is used today in the diagnosis of the acute abdominal conditions, in contrast to the complete dependence on history, physical findings, and too often exploration, which existed not much more than a decade ago. I believe that the additional information obtained through the extension of our vision through the abdominal wall has produced much more rational therapy.

Dr. French (closing). In reply to Dr. Golden's question, there was ulceration of the colon in the example shown here. In the experimental work reported by Saul Kay in the *Archives of Pathology* in April, 1954, I believe he was unable to produce the granulomas without injury to the mucosa.

I believe the answer to the question is that injury to the mucosa would be a prerequisite.

Dr. Marshak (closing). The earliest roentgen changes are difficult to evaluate.

It is impossible to state whether earlier changes different from those described exist or can be recognized as most of the patients are examined when the disease process is well established. It has been possible, however to demonstrate early changes in recurrences following side-tracking operations and these changes are similar to those noted in the intestinal segments less severely involved by regional enteritis. We have considered thickening and blunting of the folds, slight rigidity of the bowel wall and loss of the normal coiled appearance of the small bowel as the earliest roentgen changes. Occasionally peculiar irregularities of the contour, some of which appear to be submucosal, are noted in the early stages of this disease. These changes may not persist and could be due to serosal and mesenteric inflammation rather than intrinsic disease. Ulceration of the mucosa occurs after the above described changes.

THE RELATION OF THE CAPACITY OF THE HUMAN SKULL TO ITS ROENTGENOLOGICAL LENGTH

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HAAS has stated that the roentgenological length of a skull from glabella to opisthocranion is a reliable index of its size, if the skull has normal configuration.[1] The statement was based on a roentgenological investigation of live human skulls whose true capacities could not be known.

The present investigation was carried out on dry skulls with the object of relating known capacities to roentgenological lengths and of calculating the dimensions of the errors inherent in using length as an index of capacity.

METHODS

Fifty-three dry adult skulls with detachable vaults were collected at random from various departments of the University of London. No attempt was made to classify them according to sex and race. One was discarded when its walls were found to be thick and failed to define themselves sharply on the roentgenograms.

Cotton wool plugs were introduced into the superior orbital fissures, into the jugular foramina and into the foramina ovale from the outside of the skulls. The remaining apertures, with the exception of the foramina magna, were filled from the inside, and a wisp of cotton wool placed on each cribriform plate. The vaults of the skulls were applied and fixed firmly.

Lead shot of mixed sizes was then introduced into the skulls through the foramina magna. After the introduction of approximately 700 cc. each skull was shaken front down to ensure the filling of the anterior cranial fossae. As further lead was introduced it was directed and packed by finger, the filling being performed under the same tension in each skull. The contents were poured into two 1,000 cc. measuring jars, shaken and measured. The maneuver of filling and measuring was practiced until

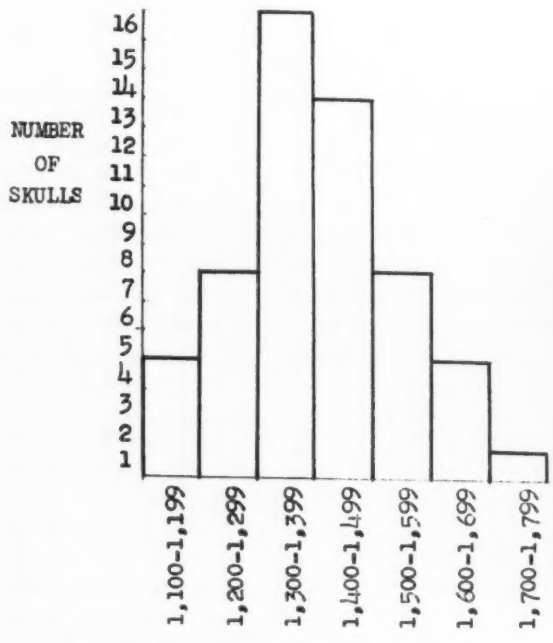

FIG. 1. Histogram of the capacities of 52 dry skulls chosen at random.

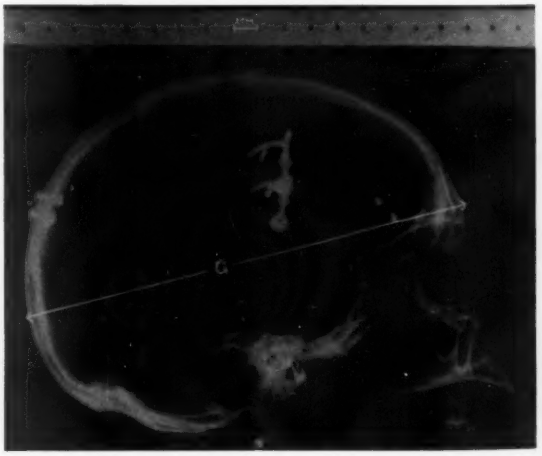

FIG. 2. Lateral roentgenogram of dry skull showing length (G) from glabella to episthocranion. A measuring bar is placed above the skull.

TABLE I

CAPACITIES (CUBIC CENTIMETERS) AND ROENTGENOLOGICAL LENGTHS (CENTIMETERS) OF 52 DRY ADULT SKULLS

Serial No. Skull	Capacity (V)	Length (G)	$\frac{V}{G}$	$G \times$ Average $\frac{V}{G}$	Departure of Col. 1 from Col. 4	$\frac{\text{Col. 5} \times 100}{V}$	Serial No. Skull	Capacity (V)	Length (G)	$\frac{V}{G}$	$G \times$ Average $\frac{V}{G}$	Departure of Col. 1 from Col. 4	$\frac{\text{Col. 5} \times 100}{V}$
	Col. 1	Col. 2	Col. 3	Col. 4	Col. 5	Col. 6		Col. 1	Col. 2	Col. 3	Col. 4	Col. 5	Col. 6
1	1,410	18.45	76.4	1,439	29	2.1	27	1,430	17.6	81.3	1,373	57	4.0
2	1,420	17.7	80.2	1,381	39	2.7	28	1,285	17.5	73.4	1,365	80	6.2
3	1,330	18.45	72.1	1,439	109	8.2	29	1,300	17.2	75.6	1,342	42	3.2
4	1,690	19.25	87.8	1,502	188	11.0	30	1,125	16.9	66.6	1,318	193	17.2
5	1,415	17.8	79.5	1,388	27	1.9	31	1,285	17.6	73.0	1,373	88	6.8
6	1,365	16.9	80.8	1,318	47	3.4	32	1,365	18.2	75.0	1,420	55	4.0
7	1,325	17.6	75.3	1,373	48	3.6	33	1,150	16.9	68.0	1,318	168	14.6
8	1,155	17.05	67.7	1,330	175	15.2	34	1,510	18.4	82.1	1,435	75	5.0
9	1,345	17.9	75.1	1,396	51	3.8	35	1,290	18.45	69.9	1,439	149	11.6
10	1,355	17.7	76.6	1,381	26	1.9	36	1,605	18.9	84.9	1,472	133	8.3
11	1,540	18.1	85.1	1,412	128	8.3	37	1,490	17.9	83.2	1,396	94	6.3
12	1,260	16.9	74.6	1,318	58	4.6	38	1,585	17.8	89.0	1,388	197	12.4
13	1,335	17.9	74.6	1,396	61	4.6	39	1,700	18.8	90.4	1,466	234	13.8
14	1,475	18.6	79.3	1,451	24	1.6	40	1,560	17.9	87.2	1,396	164	10.5
15	1,320	18.1	72.9	1,412	92	7.0	41	1,485	18.9	78.6	1,474	11	.7
16	1,590	18.05	88.1	1,408	182	11.4	42	1,300	17.35	74.9	1,353	53	4.1
17	1,460	18.0	81.1	1,404	56	3.8	43	1,200	16.9	71.0	1,318	118	9.8
18	1,450	18.5	78.4	1,443	7	.5	44	1,310	18.7	70.1	1,459	149	11.4
19	1,490	18.0	83.1	1,404	91	6.1	45	1,290	17.2	75.0	1,342	52	4.0
20	1,455	18.2	79.9	1,420	35	2.4	46	1,300	17.5	74.3	1,365	65	5.0
21	1,370	18.6	73.7	1,451	81	5.9	47	1,325	18.1	73.2	1,412	87	6.6
22	1,640	18.2	90.1	1,420	220	13.4	48	1,340	18.0	74.4	1,404	64	4.8
23	1,455	18.3	79.5	1,427	28	1.9	49	1,285	17.05	75.4	1,330	45	3.5
24	1,380	18.2	75.8	1,420	40	2.9	50	1,630	18.2	89.6	1,420	210	12.9
25	1,415	18.3	77.3	1,427	12	.8	51	1,525	17.7	86.2	1,381	144	9.4
26	1,195	17.4	68.7	1,357	162	13.6	52	1,540	18.8	81.9	1,466	74	4.8

the technique yielded results with a maximum error of 5 cc. on immediate repetition and a maximum error of 10 cc. when repeated at weekly intervals. The capacities of the 52 skulls are recorded in Table 1 and arranged as a histogram in Figure 1.

A Potter-Bucky roentgenogram was made of each skull in lateral perspective with a measuring bar, perforated at 1 cm. intervals, suspended above its midline (Fig. 2). The measurement from the glabella to the opisthocranion (G) was taken in each case and recorded in Table 1.

Table 1 contains the true capacity (V), the glabella-opisthocranion length (G) and the ratio of capacity to length (V/G) of each skull. In addition, it contains the estimated capacities of the skulls (Col. 4) obtained by multiplying the length of each by the average ratio of capacity to length. It also contains the departures of the estimated from the true capacities in cubic centimeters (Col. 5), and in percentages of the true capacities (Col. 6). The percentage departures from the true capacities are arranged histographically in Figure 3.

The graph in Figure 4 was obtained by calculating from the histogram in Figure 3

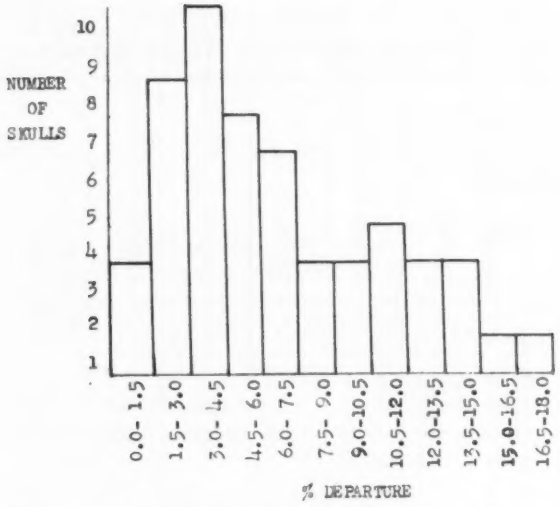

FIG. 3. Histogram of percentage departure of estimated capacities (based on length) from true capacities of 52 skulls.

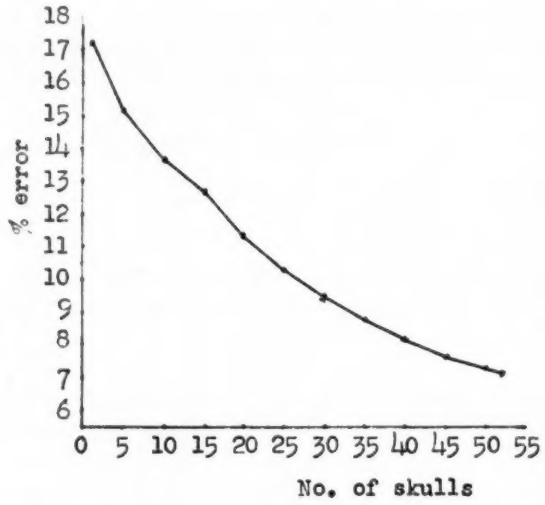

FIG. 4. Graph of maximum error in estimating skull capacity from skull length.

the maximum error introduced into the estimation of the capacity of one skull, and the maximum errors which were introduced into the estimation of the average capacities of groups of skulls varying in number up to 52.

RESULTS

Table 1 (Col. 1) shows that the true capacities of the skulls ranged from a minimum of 1,125 cc. to a maximum of 1,700 cc. The average was calculated to be 1,401 cc. Twenty-nine out of the 52 had capacities of 1,300 cc. to 1,499 cc., slightly more than half of these being below 1,400 cc. (Fig. 1). The remainder ranged equally on either side of the main group.

Table 1 (Col. 2) shows that the lengths of the 52 skulls used in the present investigation varied between a minimum of 16.9 cm. and a maximum of 19.25 cm. The average was calculated to be 17.9 cm.

Table 1 (Col. 3) shows that the ratios of skull capacities to skull lengths varied from a minimum of 66.6 to 1 to a maximum of 90.4 to 1. The average was calculated to be 78 to 1. This average ratio is the average relation between skull capacity and skull length found in the present investigation. Therefore multiplication of the length of any of the skulls by 78 offers the best method of estimating its capacity from its length.

Table 1 (Col. 6) demonstrates that the maximum error introduced into the estimation of skull capacity by multiplying skull length by 78 was 17.2 per cent, or ± 241 cc. in a 1,400 cc. skull. Figure 4 shows that, starting with this maximum error of 17.2 per cent in the estimation of the capacity of one skull, the maximum error in estimating the average capacity of groups of skulls diminishes as the number of skulls increases until finally it is of the order of 7.5 per cent when 52 skulls are used. This error is equal to ± 105 cc. in skulls with an average capacity of 1,400 cc.

DISCUSSION

Haas[1] examined 395 adult skulls of mixed sex and race and found their average roentgenological length to be 21.2 cm., with 16 per cent roentgenological enlargement. Correction for the enlargement gives a figure of 18.0 cm. The average roentgenological length obtained during the present investigation was 17.9 cm., which corresponds closely with the average obtained by Haas.

Bryce[2] states that the average capacity of adult crania of mixed races has a value of 1,400 cc., a figure which corresponds

very closely with the average of 1,401 cc. obtained in the present investigation.

Bryce[2] further states that the range of the average adult skull capacity of mixed races lies between 1,000 cc. and 1,800 cc. The range of 1,125 cc. to 1,700 cc. obtained in the present investigation is slightly less than the limits of normality, but their histographic form (Fig. 1) suggests that the skulls provide a reasonable sample of range.

In view of the close correspondence between the average roentgenological skull length obtained in the present investigation with that obtained by Haas, and of the close relationship of the capacity findings to those of previous observers (Bryce[2]), it is suggested that the skulls used in this investigation can be regarded as representative of a mixed adult population, and that what is true for them is true for adult skulls as a whole.

Hence, if the roentgenological length of any skull in centimeters from glabella to opisthocranion is known, it is suggested that its multiplication by 78 will provide the optimum estimation of its capacity in cubic centimeters. Further, it is suggested that the maximum error which can be introduced into the estimation of the capacity of a single skull by multiplying its length by 78 is approximately 17.2 per cent, and that the error in estimating the average capacity of a group of skulls by this method diminishes as the number of skulls in the group increases until the error is approximately 7.5 per cent in a group of 52.

SUMMARY

1. The capacities of 52 dry adult skulls chosen at random were obtained by measuring the volume of lead shot which was required to fill them.

2. Lateral roentgenograms of the same skulls were made and their lengths measured from glabella to opisthocranion.

3. The average ratio of the capacities (cubic centimeters) to the lengths (centimeters) was found to be 78 to 1. An estimated capacity of each skull was therefore obtained by multiplying its length by 78.

4. The greatest difference between the estimated and true capacity of any one skull was 17.2 per cent, or ±241 cc. in a 1,400 cc. skull.

5. It was calculated that the difference between the estimated and true capacities of groups of skulls diminished as the number of skulls in the groups increased until it reached 7.5 per cent when the group consisted of 52 skulls.

6. It was argued that the results obtained from the 52 skulls investigated are applicable to adult skulls in general.

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THE ROENTGEN MANIFESTATIONS OF EARLY JOINT DISEASE*

By DORIS E. PIPKIN, M.D., JACK MOSHEIN, M.D., *and* EVERETT L. PIRKEY, M.D.†
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IN VIEW of the very rapid advances in the past fifteen years in the treatment of infectious processes, it is thought to be time for radiologists to review their concepts concerning the stage of joint disease at which the diagnosis should be made. Most of the articles appearing in the medical journals, as well as the remarks made in textbooks, would lead one to believe, for example, that tuberculous arthritis is diagnosable only at the time when bone destruction has become evident. The same general feeling prevails concerning pyogenic type infection of the joints. This is not true, and with the advent of the antibiotics we are beginning to see joint infections which have run their course without any obvious bone destruction. If the radiologists are to remain of service to the clinician in these cases, it behooves them to think in terms of joint disease, infectious and aseptic in origin, before obvious bone destruction has occurred. Our paper will discuss several of the more common types of joint disease. We will divide the changes into four principal groups: (1) soft tissue changes; (2) joint space changes; (3) joint surface changes, and (4) bone changes. (See Chart 1).

Since the hip joint is the most commonly involved joint, the largest joint, and rather accurately mirrors the changes seen in other joints, it will be used as our principal example throughout this discussion.

SOFT TISSUE CHANGES

Soft tissue changes are the earliest signs of joint disease. They are the most easily

CHART I

ROENTGEN FINDINGS IN EARLY JOINT DISEASE

	Tuberculous Infections	Pyogenic Infections	Perthes' Disease	Hemophilia	Osteochondritis Dissecans
Soft tissues	Beginning atrophy of the muscles	Marked edema	Beginning atrophy of muscles	None	None
Joint space	Effusion	Effusion	Widening of the joint space	Effusion	Effusion
Joint surface	None early. Later erosions at the lateral margins	None	None	None	Punched-out area with central nidus
Bone changes	Bone atrophy. Occasionally small cystic areas are seen in the metaphysis	None	Increased density of the capital femoral epiphysis; also demineralization of the metaphysis	Bone atrophy	Bone atrophy

* Aided in part by a grant to the medical school from the Kentucky State Medical Research Commission.
The illustrations were prepared by the Department of Visual Education, University of Louisville School of Medicine.
Cases obtained from the Kosair Crippled Children Hospital and Louisville General Hospital, Louisville, Kentucky.
Read at the Annual Meeting of the Rocky Mountain Radiological Society, Denver, Colorado, August 20–22, 1953.
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overlooked changes in the interpretation of joint disease and one must constantly be aware of them. Bone pathology is usually quite visible to even the medical student and it is well for the radiologist to pay more attention to the soft tissues about joints than to the bones. They may lead to a quite early diagnosis of disease in the joint if one is aware of them and on guard as to their significance. The two principal changes that may occur in the soft tissues about any joint affliction are: (1) edema, and (2) atrophy.

Edema of the soft tissues is usually the first sign of joint inflammation, whether due to infection or trauma. It is usually very striking in children who normally have large fatty deposits between the muscle planes. The fat is more radiolucent than the adjacent muscles and gives the delineation of the muscle bundles on a properly exposed roentgenogram. Edema of the fatty tissue results in the fat having the same water density as the muscles, and therefore the intervening muscle planes are not readily visible on the roentgenogram. Edema of the soft tissue about a joint with a short history of a day or less of pain, as well as fever, chills and leukocytosis, should result in almost unequivocal diagnosis of pyogenic arthritis (Fig. 1 and 2). Edema of the soft tissues about the joint with a chronic history of pain of a more vague nature should result in a tentative diagnosis of a more chronic disease.

Fig. 1. J.F., six week old white male admitted as a feeding problem. Twelve days after admission signs of inflammation of the right hip appeared. Culture of material aspirated from the hip yielded *Staphylococcus aureus*. Note the marked soft tissue edema and swelling of the right hip as compared with the left, and the dislocation which has occurred as a result of the joint effusion. The right acetabulum is of the same configuration as the left indicating that the dislocation is not congenital.

Fig. 2. W.H., five week old infant admitted because of drainage from the left hip region. This patient had received an injection in the left hip one week after birth because of convulsions. Two weeks later he began to run a fever and swelling of the left hip was noted. This swelling was "lanced" by a private doctor and he was given penicillin. Culture of the purulent material was positive for *Staphylococcus aureus*. Note the same changes as in Figure 1, plus early destructive changes in the metaphyseal region of the left femoral neck.

The second common soft tissue change is *atrophy*. This is commonly seen in patients with a more chronic type of inflammation of the joint. The pain and other systemic symptoms are usually not sufficient in tuberculous arthritis to bring the patient to the physician until some months have elapsed after the onset of the disease. As a result, the soft tissue atrophy is quite apparent at the time of the initial examination (Fig. 3*A*). This same finding is usually quite apparent in Calvé-Legg-Perthes' disease (Fig. 7). Also here the pain and the disuse have been prolonged over a period of some months. Atrophy about the joint may also be due to primary muscle or nervous disease, such as poliomyelitis, which should be excluded in the history.

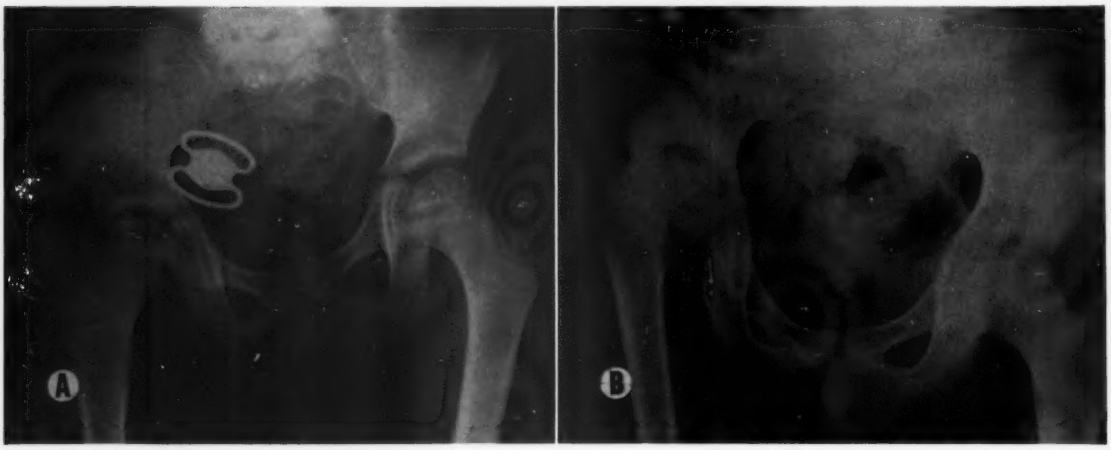

FIG. 3.(*A*) R.Mc., six year old white male who developed increasing pain in the right hip followed by a limp three months prior to entry to the hospital. On admission, culture of the joint effusion was positive for tuberculosis. The roentgenogram shows marked soft tissue atrophy of the right hip in comparison to the left; also, demineralization of the capital epiphysis and the metaphyseal region extending about one-third of the way down the femoral shaft. There are no joint surface changes. However, there are several small cystic areas seen in the acetabular region medially. (*B*) Same patient three and one-half years later with the typical tuberculous changes in the right hip.

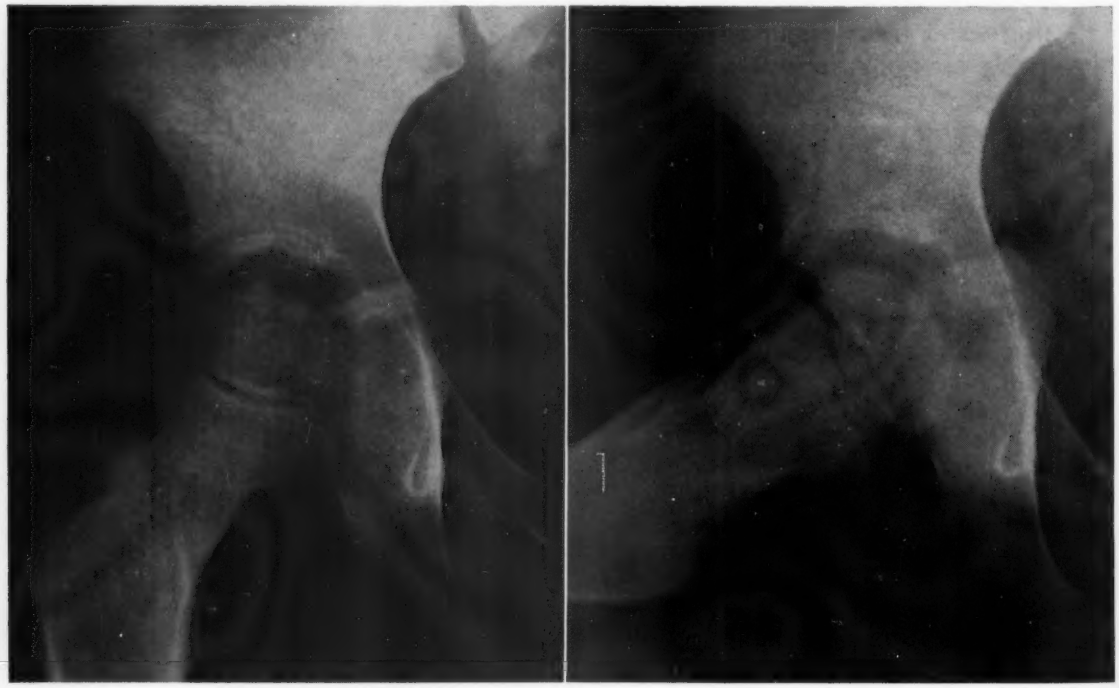

FIG. 4. G.S., five year old white male who had a history of increasing pain and spasm in both hips for one year. A biopsy of the left hip showed tuberculosis. Note the early signs of tuberculosis of the hip joint, demineralization of the femoral head, neck and shaft, effusion with widening of the joint space, an obturator sign, and small cystic changes in the acetabulum.

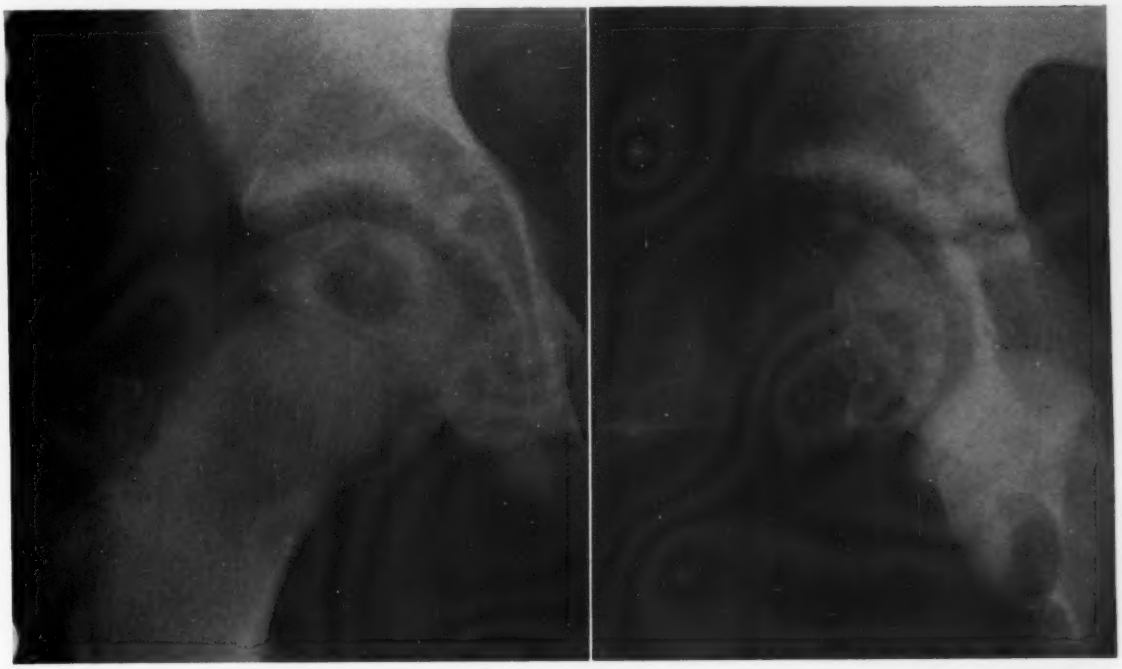

Fig. 5. T.M., white male, twelve and one-half years of age, with a history of pain in the right hip and limp for one year. The parents had noticed that the right thigh had become smaller than the left. A biopsy confirmed the diagnosis of tuberculosis of the hip. The roentgenogram shows a large cystic-like abscess in the capital epiphysis of the femur, demineralization of the bones surrounding the joint, and effusion with some widening. Compare this roentgenogram with Figure 6.

JOINT SPACE CHANGES

The joint space may present early evidence of a disease process. This usually is represented by two types of changes. One is that of widening, which results from effusion or thickening of the synovial membrane due to the disease process and the other is narrowing of the joint space, which results from atrophy of the synovia and either atrophy or destruction of the articular cartilage.

In infants under two years of age, acute infection in the hip joint commonly results in actual subluxation of the joint due to the increased intra-articular pressure and frequently these children on admission are mistakenly diagnosed as congenital dislocations of the hip (Fig. 1 and 2). However, this can readily be differentiated by studying the appearance of the acetabulum which has a more vertical type roof in the patients with congenital dislocation and is also much more shallow (Fig. 8). In early Calvé-Legg-Perthes' disease the joint space is widened medially due to swelling and thickening of the soft tissues at the attachment of the ligamentum teres to the acetabulum (Fig. 7). In joint disease due to hemorrhagic phenomena, either from trauma or actual blood pathology, such as hemophilia, the widening of the joint space is very apparent on the films (Fig. 9). The history, as well as the absence of generalized systemic symptoms, should be helpful in the differentiation between early pyogenic arthritis and hemophilic arthritis.

Narrowing of the joint space is rarely a sign of early joint disease. It may be considered as an early sign of osteoarthritis and will sometimes be the first sign in early post-traumatic arthritis of the joint. In these patients it is usually due to some traumatic change causing loss of the articular cartilage and in osteoarthritis it occurs from actual atrophy of the articular cartilage and the synovial membrane.

FIG. 6. E.F., ten year old white male who had a history of an insidious onset of a limp of the left lower extremity for one year. The limp was followed by pain in the left hip and knee. This roentgenogram shows flattening of the superior articular surface of the capital femoral epiphysis, widening of the joint space medially, and the demineralization of the metaphysis adjacent to the epiphyseal line. Compare this roentgenogram with Figure 5. This is Calvé-Legg-Perthes' disease.

FIG. 7. R.P., nine year old white male with a history of insidious onset of limp of the right lower extremity for five months, followed by pain. Physical examination revealed a limited range of motion of the hip in all directions. The capital femoral epiphysis on the right shows a marked increase in density compared to the left, due to the beginning aseptic necrosis. There is marked soft tissue atrophy about the hip joint, and marked widening of the joint space medially. This is Calvé-Legg-Perthes' disease.

JOINT SURFACE CHANGES

Changes of the surface of the joint are usually not seen in early disease. By the time they appear the disease process has made considerable progress. In certain instances, however, such as osteochondritis dissecans (Fig. 10), a punched-out area with a dense central nidus may be the first evidence of disease. Also, occasionally in aseptic necrosis, such as Calvé-Legg-Perthes' disease, flattening or irregularity of the articular surface of the femoral head is seen early (Fig. 6). In the infectious arthritides, both acute and chronic, when erosions of the articular surfaces of the joint are seen, the process has definitely passed beyond the early stages (Fig. 3*B*).

FIG. 8. G.P., six year old white male with a history of deformity of his right hip noted at the age of fifteen months when he first started to walk. He received no treatment for this. Six months prior to entry to the hospital he developed a limp of the left lower extremity, which was followed by pain in his left hip. A diagnosis was made of congenital dislocation of the right hip, and Calvé-Legg-Perthes' disease of the left hip. The roentgenogram shows the congenital dislocation of the hip on the right. On the left there is noted widening of the joint space medially, flattening of the superior articular surface, aseptic necrosis with early fragmentation of the capital epiphysis of the femur.

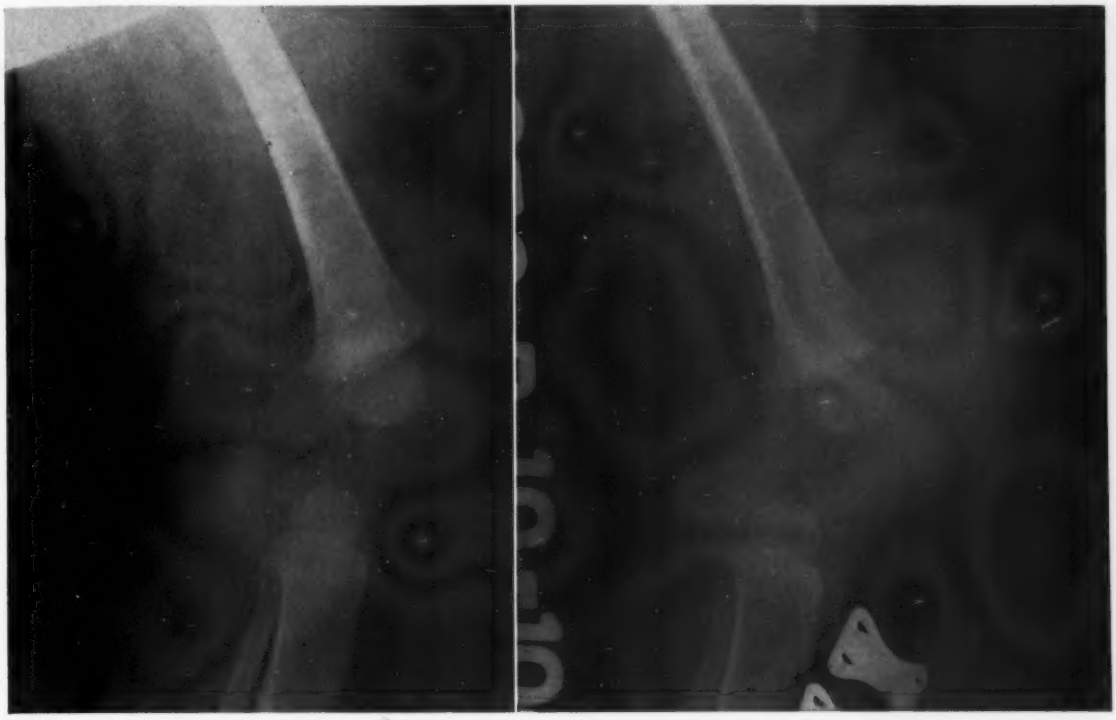

Fig. 9. T.D., one year old white male who was admitted to the hospital with the history of an injury to the right knee one month ago when he first started to walk. The swelling of the right knee has persisted since that time, and on admission to the hospital there was a 45° flexion contracture of the right knee. Blood studies revealed a bleeding time of one minute and thirty seconds, and a clotting time of thirty-eight minutes. The platelets were normal and the prothrombin was 104 per cent of normal. Repeated blood studies revealed normal bleeding time, with clotting times varying from twelve minutes to one hour and forty minutes. A diagnosis was made of hemophilia. Note the marked effusion of the left knee with the widening of the joint space in comparison with the right.

BONE CHANGES

Demineralization is the most common change in the bone seen during the early stages of joint disease. It is commonly seen in chronic joint disease, such as tuberculosis, hemophilia, and Calvé-Legg-Perthes' disease, in which cases it is partly due to the increased circulation because of the joint inflammation, as well as the changes secondary to disuse as the result of a chronic disease of the joint extending over a period of several months (Fig. 3*A* and 4). In aseptic necrosis of the joint, such as Calvé-Legg-Perthes' disease, a relative increase in density of the head of the capital epiphysis of the femur may be the first sign of disease (Fig. 7). This is accentuated by the demineralization at the junction of the epiphyseal line with the metaphysis of the neck, which is present in early stages of the disease (Fig. 6). Cystic areas in either the epiphysis, metaphysis or the adjacent acetabulum are commonly seen in early tuberculous infections of the joints, and these may be the result of an embolus of the tubercle bacillus (Fig. 3*A* and 5).

SUMMARY

The need for radiologists to consider joint disease at an early stage is presented. This has become necessary with the development of the newer antibiotics and other methods of treatment, which if instituted early, will preserve joint function and prevent the severe joint changes that used to be seen regardless of when the diagnosis was made.

Four principal types of changes are seen

FIG. 10. J.W., twenty-one year old colored male admitted with a history of occasional pain in the left knee for several years. He states that the knee will "give way" occasionally. Two days before admission he twisted his knee and since then the knee has been painful and swollen. The roentgenogram shows an osteochondritis dissecans of the lateral aspect of the medial condyle of the femur.

in early joint disease: (1) soft tissue changes, (2) joint space changes, (3) joint surface changes and (4) bone changes. These changes are discussed individually with examples of each given. A careful study of each roentgenogram for each of the changes and a summation of the findings should at least indicate to the radiologist the most likely diagnosis in each case. However, it should be pointed out that the roentgenogram will never take the place of a proper history and physical examination. Rather, the roentgen findings, plus the history and physical findings, should result in a diagnosis on the first visit.

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THE ASSOCIATION BETWEEN ANOMALOUS OSSIFICATION CENTERS IN THE HAND SKELETON, NUTRITIONAL STATUS AND RATE OF SKELETAL MATURATION IN CHILDREN FIVE TO FOURTEEN YEARS OF AGE*

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THE present report is a continuation of the series from this clinic concerning the effects of malnutrition on human skeletal status and progress.[2,3,9,12] Throughout these investigations we have been impressed by the tendency of the various ossification centers in the hand skeleton to react differently to the same intensity of nutritive failure. The skeletal maturational progress of children with chronic nutritive failure is characterized by a pronounced asymmetry.[12] The asymmetry was not corrected by milk supplements which, while accelerating the over-all rate of skeletal maturation, affected the different bone centers in varying degrees.[2] Studies of the frequency of formation of radiopaque transverse lines in the distal end of the shaft of the radius in children with and without nutritional disturbances re-emphasized the distinctiveness with which bone metabolism may be altered in the same bone center by the same precipitating factor in different children.[3] While nutritional status appeared to be independent of the tendency to scarring, it was related to the rapidity with which the transverse lines were resorbed. Regardless of nutritional status, the formation of transverse lines was noticeably reduced in the circumpubertal period.

As early as 1910, Siegert[11] reported that anomalous ossification centers in the hand skeleton, as seen in roentgenograms, may reflect growth dyscrasias. Subsequently Köhler[7] and Rochlin[10] hypothesized that such anomalies appear only when the normal process of skeletal maturation has been slowed. Since chronic under-nutrition is accompanied by asymmetry in skeletal maturation and anomalous ossification centers may be manifested in growth failure, a study was made of the frequency of anomalous centers in the hand skeleton of children with and without nutritive failure and the findings related to the speed of skeletal maturation.

MATERIALS AND METHODS

Selection of Subjects. Two hundred and sixty-six children, aged five to fourteen years, not receiving dietary supplements were chosen arbitrarily from the growth series at the Nutrition Clinic. Of these, 142 (62 boys and 80 girls) had nutritive failure§ and 124 (50 boys and 74 girls) showed neither past nor present clinical evidence of nutritional deficiencies. Each was white, of

§ "The term 'nutritive failure' perhaps means different things to different physicians. It is used in our clinic to describe a variable clinical picture and does not have a specific physiological connotation. It does not indicate why the nutrition has failed but simply that it has failed.

"Our work has convinced us that many times the physician cannot determine at once the physiologic basis of the nutritional insufficiency, and by the use of this term he can describe briefly the clinical picture without knowing the precise causation. The term has become so commonly entrenched in our clinical usage that it does not seem wise for us to restrict it or to change it.

"For the sake of clarity, the various disturbances and mechanisms which create a picture of nutritive failure we often describe separately. We can see pellagrous dermatitis or glossitis, beriberi neuritis or beriberi heart disease, scorbutic hemorrhages or gingivitis, cheilosis or the ocular symptoms of riboflavin deficiency as present symptoms arising as a response of the body to a long-continued deprivation of certain nutrients in food. Hence, nutritive failure is a more inclusive term than pellagra, beriberi, scurvy or riboflavin deficiency in that it connotes varying degrees of mixed deficiencies operating simultaneously."[12]

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Fig. 1. Supernumerary epiphysis at proximal end of metacarpal II in hand of boy with nutritive failure.

British extraction, and a native of north central Alabama.

None had known pathologies commonly associated with ossification anomalies.

The skeletal age of each child was assessed by the Todd inspectional technique[15] from an arbitrarily selected roentgenogram of the left hand. The Greulich-Pyle *Radiographic Atlas of Skeletal Development of the Hand and Wrist*[4] was used as the standard of reference. The same roentgenogram was scrutinized for structural anomalies.

Criteria for Classification of Anomalies. Shadows cast by structures on the films were classified as anomalies if they simulated ossification centers in other than anatomically normal sites; if they were abnormally oriented; if they were markedly small relative to adjacent skeletal segments and/or if they were distorted. The anomalies were categorized as: (a) Non-epiphyseal anomalies, which refer to distortions in size, shape and orientation of the bones in the phalangeal, metacarpal and carpal areas; and (b) "Epiphyseal" anomalies, which refer to centers of ossification at sites other than the location of the customary or true epiphysis. "Epiphyseal" anomalies were grouped as follows:

(1) Supernumerary center of ossification. A center of ossification occurring at the non-epiphyseal end of the shaft of a metacarpal or of the proximal or middle phalanges. Ultimately such centers unite with the shaft (Fig. 1).

(2) Pseudoepiphysis. An ossified area in the non-epiphyseal end of a metacarpal or a phalanx in which ossification, by extension from the primary center, proceeded in such manner as to leave a uni- or bilateral non-osseous notch or fissure-like hiatus in the shaft. Irrespective of whether the hiatus was uni- or bilateral, the ossified area was not classified as a pseudoepiphysis unless (a) the fissure was relatively long, or (b) a more or less dense white line extended from the medial end of the fissure toward the opposite border of the bone or toward the medial end of the opposing fissure in cases where the hiatus was bilateral. The dense white line was interpreted as indicative of nearly complete synostosis. On the first metacarpal, instead of such a line, a more radiolucent proximally curved line or groove extending from the radial to the ulnar border of the distal end of the shaft was considered evidence of a pseudoepiphysis. In some instances, a slit-like hiatus extending as a continuation of this curved line supplied confirming evidence. A notch on the radial margin of the first metacarpal practically always marked the more easily visible end of the line (Fig. 2 and 3).

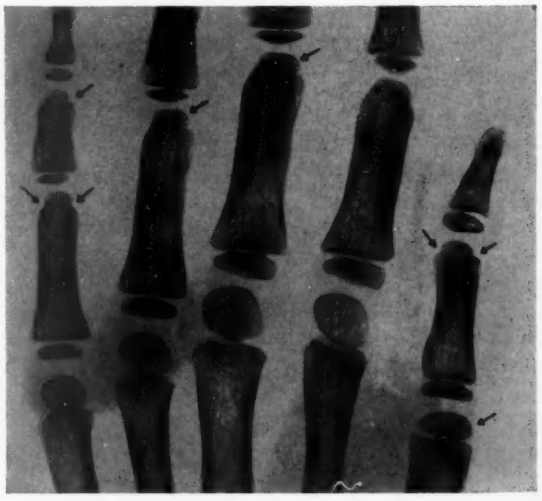

Fig. 2. Pseudoepiphyses in hand of 7 year 8 month old boy: Distal end of metacarpal 1; L- and reverse L-shaped notch on proximal phalanx of thumb, and middle phalanx of little finger; incipient rectangular or C-shaped notches on proximal phalanges 3, 4, 5

(3) Notch. A generic term comprising the following varieties many of which have been interpreted as pseudoepiphyses.[10,14]

(a) L-shaped, either normal or reversed: These occur in one stage of the growth and development of the distal end of the proximal and middle row of phalanges. They may be unilateral or bilateral and are apparently a transitory modification leading to Type b in these phalanges.

(b) Open-end short rectangle or square (occasionally resembles a C or reverse C): They are found at the distal end of the phalanges, extending roughly perpendicular to the shaft. They develop, seemingly, as the most distal portion of that part of the shaft widens to approximate its adult size. Near the proximal end of the metacarpals, either the rectangular or square type of notch may result from the peripheralward ossification of a fissure-like hiatus. (See description of pseudoepiphysis.) The angular forms may develop also when the inverted L-shaped interval at the ulnar margin of the proximal fifth of the fifth metacarpal or a similar interval at the radial margin of the second metacarpal is progressively ossified (Fig. 4).

(c) Inverted L: These occur, as just described, on the second and fifth metacarpal. When present, they are apparently a particular modification in the widening and modeling of the base of these shafts (Fig. 5).

Fig. 3. Pseudoepiphyses in hand of 7 year 11 month girl: Pseudoepiphysis on metacarpals I and II; and unilateral notch on radial border of metacarpal V.

Fig. 4. Hand of 8 year 8 month old boy showing pseudoepiphysis on metacarpal I; rectangular notch on proximo-radial border of metacarpal II; L- and reversed L-shaped notches on proximal phalanx of thumb.

(d) Re-entrant: A type of notch found at the distal end of the second metacarpal, notably on the radial border at the junction of a supernumerary epiphysis (or a pseudoepiphysis) with the shaft. This type occurs also at the distal end of the proximal phalanx of the thumb at the junction of a pseudoepiphysis with the shaft or with the pseudoepiphysis-like modeling of the distal end of the shaft (Fig. 6).

Method of Analysis. The relationship between anomalies in the hand skeleton and the rate of skeletal maturation was determined by comparing the frequency of these anomalies in children whose skeletal age equalled or exceeded their chronological age with that in children whose chronologi-

Fig. 5. Multiple anomalies in hand of girl aged 8 years 1 month: Inverted L-shaped notch on proximo-radial margin of metacarpal II and on proximo-ulnar margin of metacarpal V; brachyphalangia and hyperplastic osteitis in mesophalanx of fifth finger.

cal age exceeded skeletal age. The relationship between the anomalies and nutritional status was established by calculating their frequency in children with and without nutritive failure. These data were then subjected to further statistical analysis. In making these analyses, "epiphyseal" anomalies and non-epiphyseal anomalies were treated separately.

OBSERVATIONS

Skeletal Age Assessments. Comparison of chronological age with skeletal age as determined from the arbitrarily selected films showed that both the number of boys with skeletal age lag and the amount of retardation was greater in the group with nutritive failure than in the group without nutritive failure (Table 1). Skeletal age equalled or exceeded chronological age in 7 of the 62 boys with nutritive failure and in 15 of the 50 boys without nutritive failure. These differences were not statistically significant. The difference between the number with or without nutritive failure whose chronological age exceeded skeletal age was significant at the 5 per cent level.

In the girls, chronological age exceeded skeletal age in 79 of the 80 with nutritive failure as compared with 31 of the 74 without nutritive failure. For the former the difference was significant at the 1 per cent level, whereas for the latter the difference was not statistically significant. No significant difference was observed between the number of girls in each group whose skeletal age equalled or exceeded chronological age.

For boys and girls combined, the number whose skeletal age equalled or exceeded chronological age was significantly greater in the group without nutritive failure. Conversely, the number whose chronological age exceeded skeletal age was significantly greater in the group with nutritive failure. Both differences were significant at the 1 per cent level.

"Epiphyseal" Anomalies and Skeletal

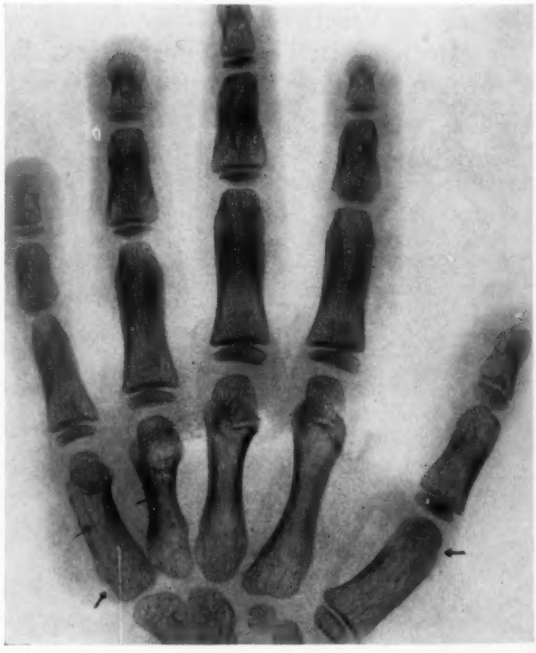

Fig. 6. Brachymetacarpalia in 5 year old girl, together with re-entrant notch on disto-radial border of metacarpal I, and inverted L-shaped notch on proximo-ulnar border of metacarpal V.

TABLE I

RELATION OF SKELETAL MATURATIONAL RATE TO NUTRITIONAL STATUS IN 266 CHILDREN

Nutritional Status	Rate of Skeletal Maturation					
	S.A. Equal or Greater than C.A.			C.A. Greater than S.A.		
	No. cases	Mean advancement (mos.)	Range (mos.)	No. cases	Mean retardation (mos.)	Range (mos.)
Boys:						
With nutritive failure	7	5.86	1–15	55	17.47	2–72
Without nutritive failure	15	7.20	0–23	35	9.49	1–24
Girls:						
With nutritive failure	1	4.0	0	79	14.62	1–36
Without nutritive failure	43	7.97	0–28	31	8.87	2–32
Total:						
With nutritive failure	8	5.63	1–15	134	15.79	1–72
Without nutritive failure	58	7.78	0–28	66	9.20	1–32

Maturational Rate. Of the 266 children, grouped on the basis of skeletal maturational rate, irrespective of nutritional status, 66 without skeletal age lag had a total of 60 "epiphyseal" anomalies; 200 with skeletal age lag had a total of 368 such anomalies. The respective means were 0.91 and 1.84; the difference between means was 5.3 times the standard error of difference (σ_D). Of the 112 boys, 22 without skeletal age lag had a total of 25 anomalies as contrasted with 194 anomalies in 90 boys with skeletal age lag. The respective means were 1.14 and 2.16; the difference was 3.17 σ_D. Among the 154 girls, 44 without skeletal age lag showed a total of 35 anomalies in contrast to a total of 174 anomalies for the 110 girls with skeletal age lag. The respective means were 0.80 and 1.58, the difference was 3.71 σ_D (Table II). All of these differences are statistically significant since they exceed σ_D 3 or more times.

Table III shows the frequency distribution of "epiphyseal" anomalies in the 266 children with and without skeletal age lag regardless of nutritional status. Of those without skeletal age lag, 36 (55 per cent) were free of anomalies; the maximum number for any child was 4. Of the children with skeletal age lag, 46 (23 per cent) were

TABLE II

STATISTICAL EVALUATION OF THE ASSOCIATION BETWEEN ANOMALOUS OSSIFICATION CENTERS AND RATE OF SKELETAL MATURATION IN 266 CHILDREN

Subject	No. Cases	No. Anomalies	Mean	σ*	Diff. in Means	No. σ_D†
Boys:						
With skeletal age lag	90	194	2.16	1.91	1.02	3.17
Without skeletal age lag	22	25	1.14	1.18		
Girls:						
With skeletal age lag	110	174	1.58	1.24	0.78	3.71
Without skeletal age lag	44	35	0.80	1.16		
Total:						
With skeletal age lag	200	368	1.84	1.42	0.93	5.3
Without skeletal age lag	66	60	0.91	1.18		

* σ = Standard deviation.
† σ_D = Standard error of difference between means.

TABLE III

COMPARISON OF THE NUMBER AND FREQUENCY OF ANOMALOUS OSSIFICATION CENTERS IN METACARPALS AND PHALANGES IN CHILDREN WHOSE SKELETAL AGE EQUALS OR EXCEEDS THEIR CHRONOLOGICAL AGE, AND IN CHILDREN WHOSE CHRONOLOGICAL AGE EXCEEDS THEIR SKELETAL AGE

No. of Anomalies	Children Whose S.A. Equals or Exceeds C.A.						Children Whose C.A. Exceeds S.A.					
	Boys		Girls		Total		Boys		Girls		Total	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0	9	41	27	61	36	55	18	20	28	25	46	23
1	5	23	6	14	11	17	19	21	24	22	43	21.5
2	5	23	5	11	10	15	21	23	33	30	54	27
3	2	9	5	11	7	11	18	20	17	15	35	17.5
4	1	5	1	2	2	3	4	4	7	6	11	5.5
5	0	0	0	0	0	0	5	6	1	0.9	6	3
6	0	0	0	0	0	0	1	1	0	0	1	0.5
7	0	0	0	0	0	0	0	0	0	0	0	0
8	0	0	0	0	0	0	4	4	0	0	4	2

free of anomalies and the maximum number for any child was 8. Only 1 girl (0.9 per cent) in this group had more than 4 anomalies as opposed to 10 boys (11.1 per cent) who exceeded this number.

The anatomical distribution and type of "epiphyseal" anomalies in the 266 children with and without skeletal age lag are shown in Table IV. The centers most frequently affected were the first, second and fifth metacarpal together with the proximal phalanx of the thumb and the middle phalanx of the little finger. Notches were far more prevalent than pseudoepiphyses. The latter were most frequently seen in the first and second metacarpals.

In the metacarpals, the frequency of anomalies in the children without skeletal age lag was 26 per cent for metacarpal I, 28 per cent for metacarpal II, and 18 per cent for metacarpal V. In the children with skeletal age lag the respective percentages were 60.5 per cent, 46.5 per cent, and 38.5 per cent. While the children with skeletal age lag had a greater frequency of anomalies in the phalanges, the differences were relatively small; none exceeded 7.5 per cent.

"Epiphyseal" Anomalies and Nutritional Status. Table V shows that the 62 boys with nutritive failure had 144 "epiphyseal" anomalies as contrasted with 75 such anomalies in the 50 boys without nutritive failure. The respective means were 2.32 and 1.50. The 80 girls with nutritive failure had 131 anomalies whereas the 74 without nutritive failure had 78. The respective means were 1.64 and 1.05. The difference between means for the groups with and without nutritive failure was significant (3.30 σ_D) only when the boys and girls were combined.

The frequency distribution of "epiphyseal" anomalies in the children with and without nutritive failure is presented in Table VI. Anomalies were seen in 112 (79 per cent) of the children with nutritive failure and in 72 (58 per cent) of the children without nutritive failure. The maximum number of anomalies observed in children with nutritive failure was 8; the maximum in children without nutritive failure was 6. Among the children with nutritive failure 9 boys and 1 girl had more than 4 anomalies whereas among those without nutritive failure this number was exceeded by only 1 boy.

The bone segments most often affected by "epiphyseal" anomalies in the children with and without nutritive failure were

TABLE IV

DISTRIBUTION OF PSEUDOEPIPHYSES AND NOTCHED SHAFTS IN METACARPALS AND PHALANGES OF 66 CHILDREN WHOSE SKELETAL AGE EQUALS OR EXCEEDS THEIR CHRONOLOGICAL AGE, AND IN 200 CHILDREN WHOSE CHRONOLOGICAL AGE EXCEEDS THEIR SKELETAL AGE

Anomaly	Children Whose S.A. Equals or Exceeds C.A.						Children Whose C.A. Exceeds S.A.					
	Boys		Girls		Total		Boys		Girls		Total	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Mtc. I (distal):												
Pseudoepiphysis	5	23	2	5	7	11	32	36	25	23	57	28.5
Bilateral notch	0	0	0	0	0	0	1	1	0	0	1	0.5
Unilateral notch	2	9	8	18	10	15	24	27	39	35	63	31.5
Total	7	32	10	23	17	26	57	63	64	58	121	60.5
Mtc. II (proximal):												
Pseudoepiphysis	1	5	3	7	4	6	10	11	4	4	14	7
Bilateral notch	1	5	0	0	1	2	2	2	0	0	2	1
Unilateral notch	7	32	6	14	13	20	39	43	38	35	77	38.5
Total	9	41	9	20	18	28	51	57	42	38	93	46.5
Mtc. V (proximal):												
Pseudoepiphysis	0	0	0	0	0	0	0	0	1	1	1	0.5
Bilateral notch	0	0	2	5	2	3	5	6	4	4	10	5
Unilateral notch	4	18	5	11	9	14	27	30	40	36	66	33
Total	4	18	7	16	11	18	32	36	45	41	77	38.5
Prox. phal. I (distal):												
Pseudoepiphysis	0	0	0	0	0	0	4	4	0	0	4	2
Bilateral notch	0	0	0	0	0	0	8	9	2	2	10	5
Unilateral notch	0	0	0	0	0	0	0	0	1	1	1	0.5
Total	0	0	0	0	0	0	12	13	3	3	15	7.5
Other prox. phal.:												
Notch (distal)	0	0	2	5	2	3	15	17	0	0	15	7.5
Mid. phal. V (distal):												
Pseudoepiphysis	0	0	1	2	1	2	2	2	1	1	3	1.5
Bilateral notch	0	0	1	2	1	2	18	20	12	11	30	15
Unilateral notch	5	23	5	11	10	15	5	6	7	6	12	6
Total	5	23	7	16	12	18	25	28	20	18	45	22.5
Other mid. phal.:												
Notch (distal)	0	0	0	0	0	0	2	2	0	0	2	1

metacarpals I, II and V together with the proximal phalanx of the thumb and the middle phalanx of the fifth finger (Table VII). Notches, which were much more numerous than pseudoepiphyses, occurred most often on metacarpals I and II. In the children with nutritive failure, phalangeal anomalies were approximately twice as frequent as in those without nutritive failure. In the children with nutritive failure the frequency of anomalies in the metacarpals was 65 per cent in metacarpal I, 49 per cent in metacarpal II, and 37 per cent in metacarpal V. The corresponding frequencies in the children without nutritive failure were 37 per cent, 33 per cent, and 29 per cent.

Non-epiphyseal Anomalies. Six types of

Table V

STATISTICAL EVALUATION OF THE ASSOCIATION BETWEEN ANOMALOUS OSSIFICATION CENTERS AND NUTRITIONAL STATUS IN 266 CHILDREN

Subject	No. Cases	No. Anomalies	Mean	σ*	Diff. in Means	No. σ_D†
Boys:						
With nutritive failure	62	144	2.32	2.09	0.82	2.49
Without nutritive failure	50	75	1.50	1.32		
Girls:						
With nutritive failure	80	131	1.64	1.26	0.59	2.95
Without nutritive failure	74	78	1.05	1.26		
Total:						
With nutritive failure	142	275	1.94	2.14	0.71	3.30
Without nutritive failure	124	153	1.23	1.31		

* σ = Standard deviation.
† σ_D = Standard error of difference between means.

non-epiphyseal anomalies were noted in the hand skeleton of the 266 children, *viz.*, brachymetacarpalia, brachyphalangia, clinodactylia, camptodactylia, notching of capitate, and separation of the styloid process of ulna.

Brachymetacarpalia, or a marked shortness in metacarpal segments, occurred in only 1 child (Fig. 6). She was in the group without nutritive failure, had disproportionately short fourth and fifth metacarpals, and was two months advanced in skeletal age.

Brachyphalangia, or a disproportionate shortness in the phalanges, was seen only in the mesophalanx of the little finger and occurred independently of or in association with notching (Fig. 7). In combination with the dwarfing, this phalanx often showed poor modeling and tubulation. Six boys and 4 girls in the group with nutritive failure had a dwarfed mesophalanx of the little finger as compared with 1 boy and 6 girls in the group without nutritive failure. Of the 17 children, 13 were retarded in skeletal age and 4 advanced.

Radialward clinodactylia involving angulation of the terminal phalanx of the

Table VI

COMPARISON OF THE NUMBER AND FREQUENCY OF ANOMALOUS OSSIFICATION CENTERS IN METACARPALS AND PHALANGES IN CHILDREN WITH AND WITHOUT NUTRITIVE FAILURE

No. of Anomalies	Children with Nutritive Failure						Children without Nutritive Failure					
	Boys		Girls		Total		Boys		Girls		Total	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0	13	21	17	21	30	21	14	28	38	51	52	42
1	11	18	20	25	31	22	13	26	10	14	23	18
2	14	23	25	31	39	27	12	24	13	18	25	20
3	12	19	12	15	24	17	8	16	10	14	18	14
4	3	5	5	6	8	6	2	4	3	4	5	4
5	5	8	1	1	6	4	0	0	0	0	0	0
6	0	0	0	0	0	0	1	2	0	0	1	1
7	0	0	0	0	0	0	0	0	0	0	0	0
8	4	6	0	0	4	3	0	0	0	0	0	0

TABLE VII

DISTRIBUTION OF PSEUDOEPIPHYSES AND NOTCHED SHAFTS IN METACARPALS AND PHALANGES OF 142 CHILDREN WITH NUTRITIVE FAILURE AND 124 CHILDREN WITHOUT NUTRITIVE FAILURE

Anomaly	Children with Nutritive Failure						Children without Nutritive Failure					
	Boys		Girls		Total		Boys		Girls		Total	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Mtc. I (distal):												
Pseudoepiphysis	18	29	22	27.5	40	28	19	38	5	7	24	19
Bilateral notch	1	2	0	0	1	0.7	0	0	0	0	0	0
Unilateral notch	20	32	31	39	51	36	6	12	16	22	22	18
Total	39	63	53	66	92	65	25	50	21	28	46	37
Mtc. II (proximal):												
Pseudoepiphysis	8	13	3	4	11	8	3	6	4	5	7	6
Bilateral notch	3	5	0	0	3	2	0	0	0	0	0	0
Unilateral notch	27	44	29	36	56	39	19	38	15	20	34	27
Total	38	61	32	40	70	49	22	44	19	26	41	33
Mtc. V (proximal):												
Pseudoepiphysis	0	0	0	0	0	0	0	0	1	1	1	0.8
Bilateral notch	4	6	2	2.5	6	4	1	2	4	5	5	4
Unilateral notch	19	31	27	34	46	32	12	24	18	24	30	24
Total	23	37	29	36	52	37	13	26	23	31	36	29
Prox. phal. I (distal):												
Pseudoepiphysis	2	3	0	0	2	1	2	4	0	0	2	1.6
Bilateral notch	8	13	2	2.5	10	7	0	0	0	0	0	0
Unilateral notch	0	0	1	1	1	0.7	0	0	0	0	0	0
Total	10	16	3	4	13	9	2	4	0	0	2	1.6
Other prox. phal.:												
Notch (distal)	13	21	0	0	13	9	2	4	2	3	4	3
Mid. phal. V (distal):												
Pseudoepiphysis	0	0	0	0	0	0	2	4	2	3	4	3
Bilateral notch	16	26	10	12	26	18	2	4	3	4	5	4
Unilateral notch	3	5	4	5	7	5	7	14	8	11	15	12
Total	19	31	14	17.5	33	23	11	22	13	18	24	19
Other mid. phal.:												
Notch (distal)	2	3	0	0	2	1	0	0	0	0	0	0

fifth finger toward the radius was noted in 1 boy and 2 girls in the group with nutritive failure and in 2 boys and 4 girls without nutritive failure (Fig. 7). Of the 9 children, 5 were advanced and 4 retarded in skeletal age.

Camptodactylia, flexion of a digit with inability to extend it except forcibly, was found in 1 boy and 1 girl with nutritive failure. In both, the fifth digit was involved, and they were retarded in skeletal age.

A relatively small C-shaped notch in the capitate, which occupied the interval between the facets for the lesser multangular and the distal end of the navicular, was found in 4 boys and 3 girls without nutritive failure and in 9 boys and 6 girls with nutritive failure (Fig. 3). Of the 22 children, 20 were retarded in skeletal age and 2 were advanced. This difference was significant at the 1 per cent level.

Separate ossification of the styloid proc-

Fig. 7. Multiple anomalies in hand of 7 year 8 month old boy: Pseudoepiphysis on metacarpal I; inverted L-shaped notch on metacarpals II and V; notches on proximal row of phalanges; brachyphalangia and poor modeling of mesophalanx of fifth finger together with notching; and clinodactyly of terminal phalanx of fifth finger.

ess of the distal ulna occurred in 2 girls with nutritive failure (Fig. 8). Both were retarded in skeletal age.

DISCUSSION

On the basis of clinical experience the etiology of pseudoepiphyses has been ascribed to disorders in one or more endocrine glands,[6,10,14] or to a very early disturbance in embryonic life possibly induced by hereditary factors.[14] The causation of pseudoepiphyses is obscured, however, by their presence in apparently normal children. Thus, Brailsford[1] found pseudoepiphyses in the hands of 8.5 per cent of 1,000 "normal" children ranging in age from birth to three years, and Posener, Walker and Weddell[8] reported pseudoepiphyses in 96 per cent of "normal" children between four and eight years of age. In Stettner's[14] series, the greatest incidence in "normal" children occurred in the seven to ten year old group, 49.5 per cent of the children being affected. Among the pathological conditions in which anomalous bone centers have been observed are encephalopathies, endocrine disturbances, congenital and familial structural defects, congenital lues, mongoloidism, and retarded growth of unspecified etiology.[1,5,6,8,10,11,14]

In none of the aforementioned studies was the attempt made to relate the frequency of pseudoepiphyses to the rate of skeletal maturation. The diagnoses of growth lag were made primarily on the basis of clinical observations. The present findings show that the frequency of "epiphyseal" anomalies is significantly greater in children with retarded skeletal maturation than in those without skeletal maturational lag. The results were significant both for the boys and girls combined and for each sex separately.

A significantly greater number of "epiphyseal" anomalies was found in children with chronic nutritive failure than in chil-

Fig. 8. Separate center of ossification for styloid process of distal epiphysis of ulna in 8 year 8 month old girl.

dren without chronic nutritive failure in this series. A statistically significant difference was not obtained when boys and girls were considered separately. The findings for the girls, however, approached significance (2.95 σ_D). The seeming discrepancy in the differences between the sexes combined and for the sexes separately may reflect sampling factors. The observation that "epiphyseal" anomalies may be associated with nutritive failure is not in accord with the report of Posener *et al.*[8] who used height, weight, muscular power and dental caries as criteria of nutritional status. The adequacy of these criteria as determinants of nutritional status is open to question.

SUMMARY AND CONCLUSIONS

The relative frequency of "epiphyseal" and non-epiphyseal anomalies was determined from a single roentgenogram of the left hand of each of 266 children ranging in age from five to fourteen years. The children were grouped on the basis of nutritional status and degree of skeletal maturation.

The mean number of "epiphyseal" anomalies in 142 children with nutritive failure was 1.94; the mean for 124 children without nutritive failure was 1.23. The mean number of "epiphyseal" anomalies for 200 children with skeletal age retardation was 1.84; the mean for 66 children with no skeletal age retardation was 0.91.

Non-epiphyseal anomalies were comparatively rare in this study.

Further analysis of the data showed:

(1) The incidence of "epiphyseal" anomalies was significantly greater in children with nutritive failure than in children without nutritive failure, regardless of the rate of skeletal maturation.

(2) The incidence of "epiphyseal" anomalies in children with skeletal age lag was significantly greater than in those without skeletal age lag regardless of nutritional status.

(3) The bones most frequently affected by "epiphyseal" anomalies in this series were: distal end of metacarpal I, proximal end of metacarpals II and V, and the distal end of the proximal phalanx of the thumb and the middle phalanx of the little finger.

(4) No significant difference in frequency was noted for the non-epiphyseal anomalies, *viz.*, brachymetacarpalia, brachyphalangia, clinodactylia, camptodactylia, and separation of the styloid process of the ulna. A statistically significant difference was observed for notching of the radial border of the capitate between children with retarded and with advanced skeletal age. The difference was not significant on the basis of nutritional status.

It is suggested that when "epiphyseal" anomalies are found in roentgenograms of the hand skeleton, the child should be examined for growth failure of endogenous or exogenous origin.

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PHYSIOLOGIC VERTEBRAL LIGAMENTOUS CALCIFICATION: AN AGING PROCESS*

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CALCIFICATION of the longitudinal spinal ligaments is a frequent finding in roentgenologic examination of the spinal column. When such calcification is localized, it is usually regarded as being a rather nonspecific, late reaction to trauma or infection; when it is more or less generalized, the presence of a more diffuse inflammatory process, such as rheumatoid spondylitis, may be suggested.

It is the purpose of this paper to describe another condition to be considered in such instances. The condition in question may be referred to as "physiologic ligamentous calcification"; it must be distinguished especially from rheumatoid spondylitis. This differentiation has not been mentioned in most presentations on the roentgenologic diagnosis of rheumatoid spondylitis.

Physiologic ligamentous calcification or ossification is associated with the decreased mobility of the spinal column that occurs with advancing age and is found in patients who have no symptoms or evidence suggestive of rheumatoid spondylitis. In a strict sense, calcification of soft tissues is not physiologic, but since it is apparently a secondary reaction to the normal changes associated with advancing age, the term "physiologic" is used to distinguish it from certain pathologic conditions in which vertebral ligamentous calcification may be found.

REVIEW OF LITERATURE

The available medical literature contains three references to this type of ligamentous calcification. The first is a description by Oppenheimer, in 1942, of 18 patients who exhibited this phenomenon. Seventeen of his patients were more than fifty-five years of age, whereas the age of the other patient was not stated. Calcification or ossification of the longitudinal spinal ligaments was present in all of his patients, unaccompanied by evidence of abnormality in the vertebral bodies, intervertebral discs or apophyseal joints. No signs or symptoms suggested the presence of disease of the spinal column. Oppenheimer described the calcified anterior longitudinal ligament as attached to the surfaces of the vertebrae but distinct from them; he said it appeared "as though it had grown too long in comparison with the bones it connected." He made no mention of the sacroiliac joints but emphasized that this condition was distinguished from Marie-Strümpell disease (rheumatoid spondylitis) by the absence of lesions of the apophyseal joints and of clinical manifestations. Oppenheimer declared that he was not describing a disease entity or a type of arthritis but a physiologic phenomenon secondary to the decreased mobility that goes with advancing age.

The other two papers have appeared in

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the literature more recently and were written by Forestier and Rotès-Querol[4,5] concerning a condition which they refer to as "senile ankylosing hyperostosis." Their description of this condition suggests that it is similar to the phenomenon described in our paper but in some respects it appears to be different. They presented 9 cases in men whose ages ranged from fifty to seventy-three (average = 65) and they described the clinical, roentgenologic and pathologic findings in some detail. Roentgenograms of the spinal column in their patients showed a continuous layer of bone anterior to and distinct from the vertebral bodies in the thoracic region. The bony layer was not continuous in the more mobile cervical and lumbar regions. The sacroiliac joints were normal in every instance.

Necropsy studies were available in 2 of their cases. The bony layer was found to be on the right anterolateral side of the spinal column in both cases; it was composed of spongiosa covered with dense cortical bone. The anterior longitudinal ligament was normal and easily detached from the bony production beneath it.

Forestier and Rotès-Querol were of the opinion that this affection of the back is different from ankylosing spondylitis, or rheumatoid spondylitis, and must be differentiated from it. It cannot be stated definitely at this time whether or not the condition they have described is the same as physiologic ligamentous calcification.

The literature also contains references to several cases presented as examples of "Bechterew's disease" or ankylosing spondylitis but which, in the light of our recent studies, appear more likely to be examples of physiologic ligamentous calcification. These include Cases 3, 8 and 9 of Schlayer and Figures 8, 9 and 10 of Simmonds.

MATERIALS AND METHODS

All patients included in the present study were seen at the Mayo Clinic between 1946 and 1951, inclusive. The basis for the inclusion of patients in this study was the finding on roentgenologic examination of the spinal column of evidence suggestive of physiologic ligamentous calcification. All available roentgenograms were reviewed in association with the clinical records in order to correlate the roentgenologic findings with any symptoms or physical findings that may have been present and to determine, if possible, the cause of the ligamentous calcification seen in each case. A special effort was made to determine if rheumatoid spondylitis could have been the underlying cause of the ligamentous calcification in any of the cases.

Fifty-three cases were found acceptable as examples of physiologic ligamentous calcification. This number by no means indicates the actual incidence of the condition, inasmuch as the significance of the finding was not recognized by all radiologists interpreting roentgenograms of the spinal column during the early years covered by this study. It may be assumed, however, from the fact that 53 cases can be presented, that the condition is not uncommon. No opportunity to supplement clinical and roentgenologic information with pathologic material presented itself during the course of this study.

RESULTS

These 53 patients who had calcification of the longitudinal spinal ligaments were divided arbitrarily into two groups. Group 1 consisted of patients who had had no symptoms referable to the back. Group 2 consisted of those patients who had had symptoms referable to the back but in whom a diagnosis of rheumatoid spondylitis could not be made.

Group 1. This group was composed of 21 patients in whom no symptoms referable to the back were present at the time of examination. There were 12 men and 9 women. Their ages ranged from forty-two to seventy-seven years, the average being 65.0 years. Only 1 patient was less than fifty years of age at the time of examination.

Nineteen patients gave no history of pain or stiffness at any time, whereas 2 had experienced pain ten and fifteen years ago, respectively, after injuries. Although none

TABLE I

REGIONS OF THE SPINAL COLUMN EXAMINED ROENTGENOLOGICALLY AND THOSE EXHIBITING LIGAMENTOUS CALCIFICATION IN GROUP I (21 PATIENTS)*

Region of Spinal Column	Examined	Involved	Other Regions of Spinal Column	Examined	Involved
Cervical	7		Thoracic	4	5†
			Lumbar	4	2
			Thoracic and lumbar	2	1
		6	Thoracic	4	4
			Lumbar	3	1
			Thoracic and lumbar	2	1
Thoracic	15		Cervical	4	4
			Lumbar	10	1
			Cervical and lumbar	2	1
		16†	Cervical	5	4
			Lumbar	12	2
			Cervical and lumbar	2	1
Lumbar	15		Cervical	4	3
			Thoracic	10	10
			Cervical and thoracic	2	2
		5	Cervical	2	1
			Thoracic	1	1
			Cervical and thoracic	1	1

* Key to the table: In the top horizontal section, note that 7 of the 21 patients in Group 1 had roentgenologic examination of the cervical portion of the spinal column and ligamentous calcification was found in 6. Of the 7 who had such examination, the lumbar portion of the spinal column was also examined in 4 patients and found to be involved by ligamentous calcification in 2. To ascertain if the cervical and lumbar portions of the spinal column were involved in the same patients, it is necessary to look in the next horizontal division, which deals with the 6 patients who had ligamentous calcification in the cervical region. For example, of the 6 patients in this group, 2 also had examination of the remainder of the spinal column. The entire spinal column was found to be involved with ligamentous calcification in 1 patient. It is also seen that data on this same patient appear in each of the three major divisions of the chart.

† In 1 patient, involvement of the lower thoracic portion of the spinal column was seen on a roentgenogram of the lumbar region, but roentgenograms of the entire thoracic region of the spinal column were not made.

of these patients complained of stiffness, 4 patients were found to have limitation of motion of the spinal column that was more pronounced than normal for persons of this age group.

The erythrocytic sedimentation rates ranged from 8 to 65 mm. during the first hour (Westergren method) in the 15 patients in whom rates were determined. Rates were normal in 7 and increased in 8 (more than 20 mm.). Unassociated diseases found in this latter group included carcinoma of the breast with metastasis (3), diabetes, myxedema, gastric polyps, anemia, prostatic hypertrophy, asthmatic bronchitis with emphysema and osteoarthritis of the hip.

Roentgenologic Findings. All patients in this group were examined roentgenologically for some reason other than symptoms referable to the back. The parts of the spinal column examined and the regions showing ligamentous calcification are shown in Table 1.

Group 2. This group was composed of 32 patients who had noted symptoms referable to the back but in whom a diagnosis of rheumatoid spondylitis could not be made. There were 24 men and 8 women, whose ages ranged from fifty-one to seventy-eight years, the average being 62.5 years.

The symptoms referable to the back were pain and stiffness; their character varied considerably. The stiffness was usually

TABLE II

AGE AT ONSET OF SYMPTOMS IN PATIENTS HAVING COMPLAINTS REFERABLE TO THE BACK (GROUP 2—32 PATIENTS)

Age of Onset, Years	Cases
30–39	2
40–49	2
50–59	11
60–69	11
70–79	2
Total	28*
Average age at onset, years	57.5

* Age of onset not given in 4 cases.

TABLE III

DURATION OF SYMPTOMS IN PATIENTS HAVING COMPLAINTS REFERABLE TO THE BACK (GROUP 2—32 PATIENTS)

Duration of Symptoms, Years	Cases
Less than 1	7
1	7
2	5
3–9	3
10–30	6
Total	28*
Average duration, months	59.0

* No duration given in 4 cases.

temporary, occurring characteristically in the morning when the patient arose and being relieved by mobilization. The pain described by most patients was vague, minimal and usually sporadic. Many described the pain as a dull ache or soreness that occurred after standing or working or when they were tired or nervous. These symptoms generally bore only a superficial resemblance to those attributed to the

TABLE IV

REGIONS OF THE SPINAL COLUMN EXAMINED ROENTGENOLOGICALLY AND THOSE EXHIBITING LIGAMENTOUS CALCIFICATION IN GROUP 2 (32 PATIENTS)*

Region of Spinal Column	Examined	Involved	Other Regions of Spinal Column	Examined	Involved
Cervical	12		Thoracic	11	12†
			Lumbar	10	6
			Thoracic and lumbar	9	6
		6	Thoracic	6	6
			Lumbar	4	3
			Thoracic and lumbar	4	3
Thoracic	23		Cervical	11	6
			Lumbar	19	11
			Cervical and lumbar	9	3
		27†	Cervical	12	6
			Lumbar	23	13
			Cervical and lumbar	9	3
Lumbar	28		Cervical	10	4
			Thoracic	19	23†
			Cervical and thoracic	9	4
		18	Cervical	6	3
			Thoracic	11	13†
			Cervical and thoracic	6	3

* See key to Table 1.

† Four patients had ligamentous calcification of the lower thoracic region seen on roentgenograms of the lumbar portion of the spinal column.

Table V

ROENTGENOLOGIC FINDINGS IN 53 PATIENTS WHO HAD PHYSIOLOGIC VERTEBRAL LIGAMENTOUS CALCIFICATION

	Group 1 (21 patients)	Group 2 (32 patients)	Total
Region of spinal column involved			
Cervical	6	6	12
Thoracic	16	27	43
Lumbar	5	18	23
Sacroiliac joints			
Negative	14	24	38
Senile obliteration	1	4	5
Not examined roentgenologically	6	4	10
Apophyseal joints			
Negative (in anteroposterior lumbar projections)	6	17	23
Indeterminate (in views available)	15	15	30
Costovertebral joints			
Negative	14	26	40
Indeterminate (in views available)	7	6	13
Osteophytes			
Present	18	25	43
Absent	3	7	10
Osteoporosis			
Present	1	0	1
Absent	20	32	52

fibrositic component of rheumatoid spondylitis.

The duration of symptoms was given for 28 of the 32 patients; it ranged from three days to thirty years. The duration of symptoms and the age of the patient at onset are summarized in Tables II and III. The average duration of symptoms before admission to the clinic was 59.0 months, and the average age at the onset of symptoms referable to the back was 57.5 years. It will be noted that the onset of symptoms was before the age of fifty in only 4 of the 28 patients for whom data were given. This is an older age of onset than usually is noted in rheumatoid spondylitis.[7,8]

Anemia was present in only 2 patients. The value for hemoglobin in 1 of these patients, who had a biliary fistula, was 10 gm. per 100 cc. of blood; the other patient, who had portal cirrhosis and hypochromic anemia, had a value of 5.8 gm. The erythrocytic sedimentation rates ranged from 1 to 120 mm. per hour. The rate was normal in 13 patients and increased in 12. Unassociated diseases found in this latter group included carcinoma with metastasis (3), active pulmonary tuberculosis, portal cirrhosis, anemia, pneumoconiosis, chronic cholecystitis with biliary fistula, pelvic tumor, benign prostatic hypertrophy and migrainous headaches. The sedimentation rate was not determined in the other 7 patients.

Roentgenologic Findings. The reason for roentgenologic examination in this group was pain or stiffness of the spinal column in 27 of the 32 patients, the other 5 being examined for unrelated conditions. The regions of the spinal column that were examined roentgenologically and those that showed ligamentous calcification are summarized in Table IV.

The roentgenologic findings in both Groups 1 and 2 are summarized in Table V. The occasional finding of senile degenerative changes in the sacroiliac joints was not at all surprising in view of the age of the patients included in this study. The only roentgenologic finding in this series that was suggestive of rheumatoid spondylitis was the presence of vertebral ligamentous calcification. However, this diagnosis was not supported by clinical, laboratory or other roentgenologic evidence.

REPORT OF CASES

Case 1. A man, aged sixty-one, came to the clinic because of several attacks of diarrhea within the preceding six months. He had noted stiffness of the lumbar portion of the spinal column, the duration of which was not stated, but had had no back pain at any time. No history of trauma could be elicited.

Examination disclosed grade 3 limitation of motion of the lumbar portion of the spinal column in all directions, on the basis of 0 being normal motion and 4 being complete limitation.

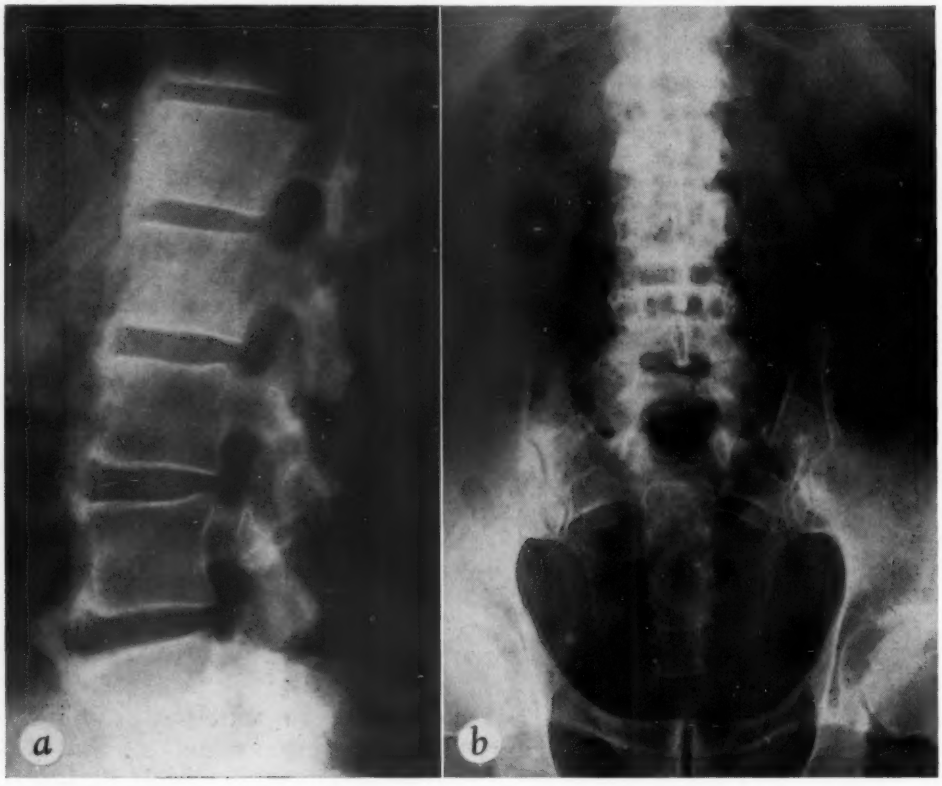

FIG. 1. Case I. *a*. Lateral view of lumbar portion of spinal column, showing a thick, calcified ligament attached to, but distinct from, the anterior surfaces of the vertebrae. The intervertebral spaces are normal. *b*. Anteroposterior view, showing normal sacroiliac joints.

Values for hemoglobin and leukocytes were within normal limits. The erythrocytic sedimentation rate was 22 mm. Roentgenologic examination of the stomach, small intestine and colon showed no abnormalities.

Roentgenograms of the lumbar portion of the spinal column disclosed a thick band of calcified ligament attached to the anterior surfaces of the lumbar vertebrae (Fig. 1*a*). The sacroiliac and lower costovertebral joints appeared to be normal (Fig. 1*b*); the apophyseal joints were not seen. The clinical, laboratory and roentgenologic findings supported the diagnosis of physiologic ligamentous calcification.

CASE II. A stock farmer, aged seventy, came to the clinic two months after an automobile accident. He was under the impression that he had received fractures of the second, third and fifth cervical vertebrae and of some ribs. His chief complaint on admission was tremor of the left hand and leg; this tremor was caused by Parkinson's disease. He complained of stiffness of the spinal column but at no time had he noted pain referable to the back, except after the recent injury.

No residual evidence of fractures of the cervical vertebrae could be demonstrated roentgenologically at the time of examination. There was grade 4 limitation of motion of the entire spinal column. Values for hemoglobin, leukocytes and erythrocytic sedimentation rate were within normal limits.

Roentgenologic examination disclosed the entire spinal column to be involved with ligamentous calcification (Fig. 2). The sacroiliac joints showed some senile narrowing. The apophyseal joints were seen in the anteroposterior projection only and showed nothing abnormal; the costovertebral joints were not adequately seen.

The presence of rheumatoid spondylitis was suspected, apparently on the basis of limitation of motion of the spinal column and the roentgenologic finding of extensive ligamentous calcification. On further review, however, the

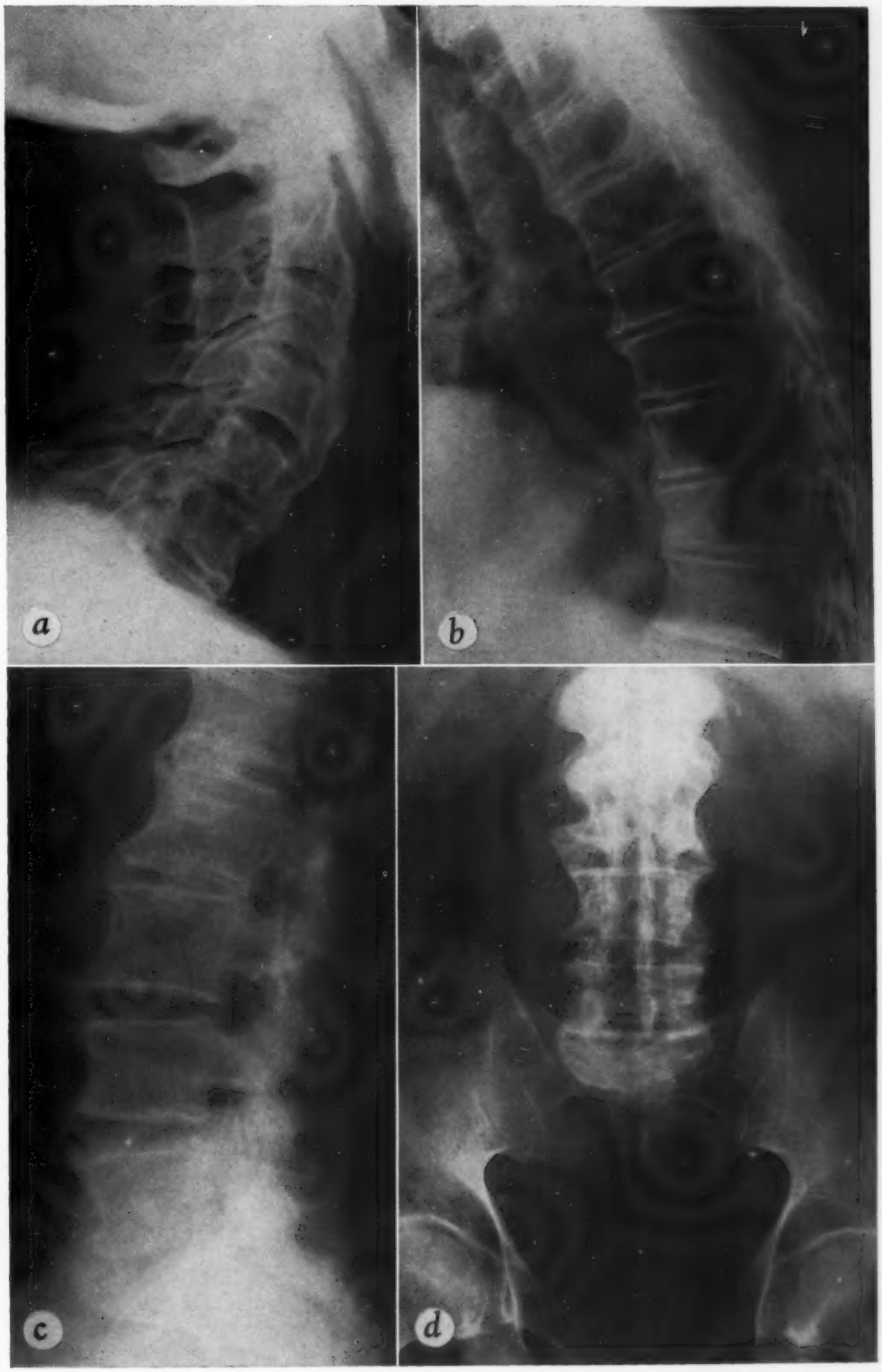

Fig. 2. Case II. *a*. Lateral view of cervical portion of spinal column, showing a bony plate 1 cm. in thickness attached to the anterior surfaces of the vertebral bodies. It has the appearance of spongy bone. *b*. Lateral

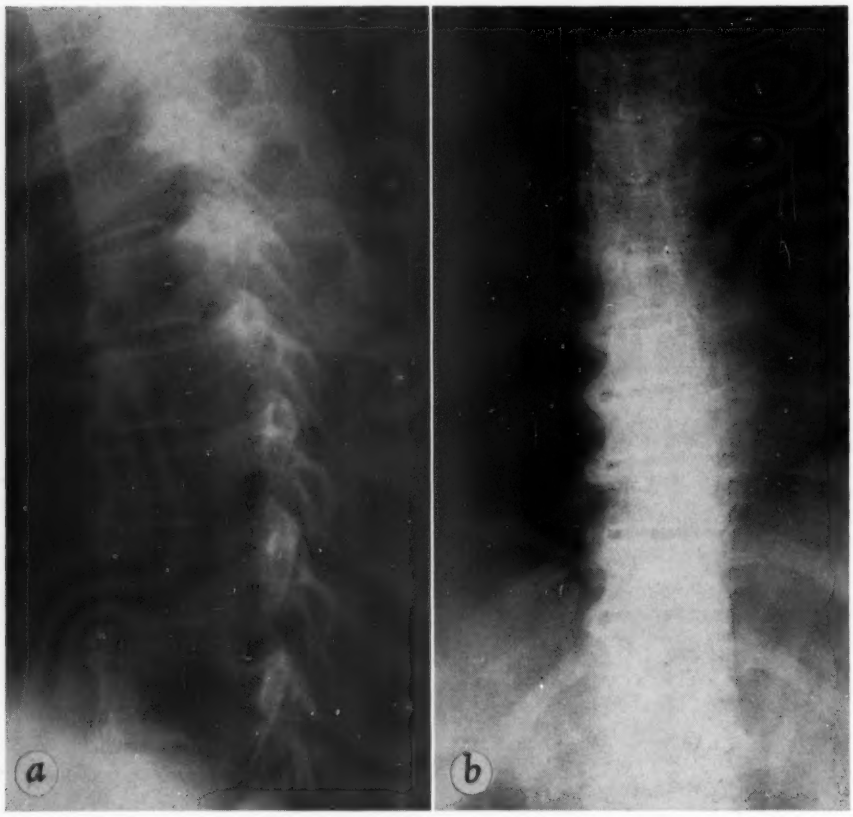

FIG. 3. Case III. *a*. Lateral view of thoracic portion of spinal column, showing calcification of the anterior longitudinal spinal ligament. It is homogeneous and distinct from the anterior margins of the vertebrae. *b*. Anteroposterior view of the thoracic portion of the spinal column, showing thick, tortuous and calcified lateral longitudinal ligaments. There is an appearance of slackness, as if the ligaments were relatively longer than the distance they span. The costovertebral joints appear normal.

history and symptoms appeared to be attributable less to rheumatoid spondylitis than to physiologic ligamentous calcification.

CASE III. A woman, aged sixty-two, came to the clinic with multiple complaints but with no symptoms referable to the back. She complained chiefly of pain in the anterior part of the thorax of thirty-five years' duration and described it as extending to the left shoulder blade. Similar pain was also noted on the right side. No history of trauma was elicited. A nodular goiter had been present for forty-five years but had produced no symptoms of toxicity. Gastrointestinal gas, bloating and belching had been experienced for forty years. She was obese, being 65 inches tall and weighing 199 pounds.

Values for hemoglobin, leukocytes and erythrocytic sedimentation rate were all within normal limits. Roentgenologic examinations revealed a normally functioning gallbladder and a duodenal ulcer.

Roentgenograms of the thoracic portion of the spinal column showed thick, tortuous and calcified anterior and lateral longitudinal ligaments in this region (Fig. 3). The costovertebral joints appeared to be normal; the apophyseal joints were not seen in these projections. Roentgenograms were not made of the sacroiliac joints nor of the remainder of the spinal column. The available evidence, however, suggested

view of thoracic portion of spinal column, showing calcification of the anterior longitudinal ligament. *c*. Lateral projection of lumbar portion of spinal column, showing extreme thickening of the anterior longitudinal ligament. *d*. Anteroposterior view, showing pronounced thickening and calcification of the lateral longitudinal spinal ligaments. This thickening is especially prominent at the level of the intervertebral spaces, giving a tortuous appearance to the ligaments. The apophyseal joints are normal and the sacroiliac joints show some senile narrowing.

that this patient had physiologic ligamentous calcification of the thoracic portion of the spinal column.

COMMENT

Features of Physiologic Ligamentous Calcification. The phenomenon of vertebral ligamentous calcification in the absence of other evidence of disease of the spinal column is characterized by several distinct features. The patients are almost invariably more than fifty years of age when physiologic ligamentous calcification is found and many have had no symptoms referable to the back. When symptoms referable to the back are present, the age of the patient at the onset of these symptoms is usually also more than fifty years. When present, the symptoms referable to the back are generally mild. Many patients show limitation of motion of the region of the spinal column that is involved but this may not be greater than would be expected in many persons of this age group. Anemia or leukocytosis is absent and the erythrocytic sedimentation rate is normal unless the presence of unassociated diseases causes an increase.

Roentgenologically, the region involved by ligamentous calcification varies in extent from a distance of one or two vertebral interspaces to the entire length of the spinal column. The thoracic portion is the most frequent site of involvement, and this region of the spinal column almost always is affected if ligamentous calcification is seen in the cervical and lumbar regions. The calcified ligament as seen on roentgenograms often reaches a thickness of 5 to 10 mm. (Fig. 1*a* and 2*a*) and frequently becomes so densely calcified as to indicate that the ligament has become ossified. It frequently appears "flabby" and as if it were too long for the intervertebral space it must cover (Fig. 2*d* and 3*b*). This appearance of looseness is especially pronounced at the intervertebral spaces. The tortuous, calcified ligament is usually homogeneous in density and is distinct from the anterior margins of the vertebrae to which it is attached (Fig. 1*a* and 3*a*). The vertebrae are of normal density and frequently show hypertrophic changes, as would be expected in patients more than fifty years of age. It is also to be expected that narrowing or partial obliteration of the sacroiliac joints would be found in some patients in this age group, but the sacroiliac joints appear normal in the great majority of patients. The apophyseal and costovertebral joints are also normal.

Differential Diagnosis of Physiologic Ligamentous Calcification. Rheumatoid spondylitis can be confused with physiologic ligamentous calcification because ligamentous calcification may be common to both. Hence, it is important to consider the differential features of these two conditions. The advanced age of the patients in whom physiologic ligamentous calcification is found, the relatively late age of onset of symptoms, if any symptoms are present, the comparative lack of significant symptoms relative to the back, the normal values for hemoglobin, leukocytes and erythrocytic sedimentation rates, the absence of roentgenologic evidence of inflammatory changes in the sacroiliac, apophyseal and costovertebral joints and the thickness and tortuous appearance of the calcified ligaments all help to distinguish this physiologic phenomenon from the pathologic process of rheumatoid spondylitis. The characteristic features of the latter condition have been adequately described by several investigators.[1–3,7–9]

The only other established condition with which this physiologic phenomenon is likely to be confused is osteoarthritis of the spinal column. Confusion can be attributed best to the fact that both entities occur in older patients and that symptoms are frequently absent or relatively mild in both. The roentgenologic appearances, however, are usually different; osteophytes and calcified ligaments generally are easily distinguishable.[9] It must be remembered, however, that ligamentous calcification and osteoarthritic changes may coexist in the same patient.

As already indicated, senile ankylosing hyperostosis, as described by Forestier and Rotès-Querol, may or may not be the same condition as physiologic vertebral ligamentous calcification.

Theory of Pathogenesis of Physiologic Ligamentous Calcification. The tortuous appearance of the calcified longitudinal ligaments suggests that they have not been under tension. With limitation of spinal motion in older patients, the spinal ligaments may be subjected to less than the usual degree of mechanical stresses. It appears probable that the degenerative changes that would occur inevitably in such ligaments might be followed by calcification and ossification unless there were some factors present to prevent such a development. The facts that a relative decrease in mobility of the spinal column occurs with advancing age and that physiologic ligamentous calcification is found in comparatively older patients suggest that decreased mobility precedes rather than follows ligamentous calcification. It is presumed that physiologic ligamentous calcification may be a result of this relative immobility of the spinal column.

Histories of trauma were given by only 14 of the 53 patients (26.4 per cent) in the entire series. This factor generally did not appear to be especially related to the finding of physiologic ligamentous calcification, inasmuch as the injuries either had occurred many years before and were not serious or were so recent that the ligamentous changes must have been present at the time of the trauma. Likewise, no correlation could be established between type of occupation and the occurrence of this phenomenon.

SUMMARY

When vertebral ligamentous calcification is encountered on roentgenologic examination of the spinal column, one should consider physiologic ligamentous calcification in the differential diagnosis. This condition was found to be the most plausible explanation for the finding of vertebral ligamentous calcification in 53 patients seen at the Mayo Clinic and included in this report. Few other reports of patients with similar or somewhat comparable conditions have been published.

Rheumatoid spondylitis is the disease with which physiologic ligamentous calcification is most likely to be confused, because ligamentous calcification may be common to both. Physiologic ligamentous calcification is suggested by the older age of the patients in whom it is found, the absence of symptoms or the relatively late age at which onset of symptoms occurs, the comparatively mild nature of symptoms relative to the back, if any symptoms are noted, the normal values for hemoglobin, leukocytes and erythrocytic sedimentation rates and the general absence of roentgenographic evidence of inflammatory changes in the sacroiliac, apophyseal and costovertebral joints.

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DYSPLASIA EPIPHYSALIS MULTIPLEX*

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JANSEN, in 1934, presented a detailed description, including biopsy, of a single case of an unusual skeletal disorder.[7] His thirteen year old, mentally defective patient showed deformity and marked limitation of motion of all the joints. Roentgenological studies demonstrated that the development of all the epiphyses was "insufficient," leading directly to joint malformation. Even the vertebral epiphyses were involved. The biopsy, taken from the region of the knee, showed delayed differentiation of cells and retarded ossification. Ample cartilaginous intercellular substance was present but calcification was quite irregular. The disease was classified as an atypical achondroplasia, but the author goes on to state that it might be better described as an "epiphyseal dysostosis." While descriptions of suspiciously similar disorders have been presented previously, these have been quite incomplete and consequently attracted little attention.[1,2,3]

Shortly after Jansen's study, Fairbank,[4] using the nomenclature dysplasia epiphysalis multiplex, described additional cases of what is apparently the same disease and added to the list of identifying features of the syndrome the presence of dwarfism and stubby digits. Since that time a total of 34 cases has been accumulated. Most of these have appeared in the British literature and have been presented by Fairbank.[5,6,10] The name, dysplasia epiphysalis multiplex, now appears to be generally accepted.

It is the purpose of this paper to present additional examples of this syndrome.

CASE REPORTS

CASE I. J.R., female, aged eleven. This patient is the younger sister of Case II and the daughter of Case III. She was a normal, full term infant whose subsequent growth and development have not been remarkable. At the present time, she enjoys normal activity and has no complaints referable to any bodily system. The patient's parents have long noted she was rather knock kneed.

Physical Examination. Height, 51 inches; weight, 63 pounds. The general appearance was that of an alert, healthy, prepubertal female. On walking she showed a duck-like gait with definite widening of the base of ambulation. Examination was not otherwise remarkable. No crepitation, limitation of motion about the joints or abnormality of muscular development was noted. Neurological examination was negative. Routine laboratory investigations were not unusual.

Roentgenological Examination. The most important studies are shown in Figures 1, 2, 3, and 4. The epiphyses of the hip, ankle, knee and wrist joints are bilaterally and symmetrically involved. All show variations of essentially the same defects; namely, deformity of outline, and irregularity of ossification. The deformity appears to be partially due to moulding and in part due to absence or failure of development of portions of the epiphysis itself. The density and trabeculation of the ossified portions of the epiphyses and of the diaphyseal segments of the long bones are within normal limits. Additional films, not presented, show the shoulders and elbows to be less markedly involved. The spine, phalanges and skull are not unusual in any respect. The configuration of the carpal bones and the epiphyses of the metacarpals are abnormal. As judged from the wrist, skeletal maturation is delayed.

CASE II. S.R., female, aged twelve. This patient was a normal, full term infant delivered by means of forceps but without incident. Her general growth and development were normal. She walked at ten months of age. At the age of sixteen months, it was first noted that she had a peculiar gait. This was characterized as waddling and with a wide base. However, she continued to develop normally and had no significant difficulty until the age of seven when she

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FIG. 1. Case 1. Anteroposterior roentgenogram of the pelvis.

first experienced pain on walking. Since that time there has been a slow but steady and progressive increase in symptomatology. At the present time she complains of definite limitation of movement about the hip joints. She experiences difficulty in climbing stairs and is unable to arise from the recumbent position without help. Within the immediate past she has developed frequent episodes of pain involving both knees.

Physical Examination. Height, 56 inches; weight, 83 pounds. Inspection showed a well developed and nourished young female of typical pubertal appearance. The gait was noted to be duck-like. There was marked genu valgum associated with angulation of the tibiae bilaterally at the ankle. No muscular atrophy or significant loss of muscle strength was observed. Neurological examination was not remarkable. Examination of the joints showed limitation of motion about the shoulders, elbows and especially prominent in the hip joints. Routine laboratory studies were unremarkable.

Roentgenological Examination. Essentially complete studies of the bones and joints were obtained. Only two films (Fig. 5 and 6) are reproduced. The findings here indicate a somewhat later stage than that seen in the younger sister. The upper femoral epiphyses are now almost completely and uniformly ossified but they remain markedly irregular in outline. Coadaptive changes in the acetabula are already apparent. Trabecular architecture and density do not appear unusual. The changes in the hands and wrists are similar, but somewhat more marked than those present in the younger

FIG. 2. Case 1 Anteroposterior and lateral roentgenograms of the right knee

FIG. 3. Case 1. Anteroposterior and lateral roentgenograms of the left ankle.

FIG. 4. Case 1. Anteroposterior roentgenograms of both wrists.

FIG. 5. Case II. Anteroposterior roentgenograms of both wrists.

sister. The configuration of the carpal bones is unusual. The radial, ulnar and metacarpal epiphyses are abnormal. Other films show ankles, knees, elbows, and shoulders to be similarly involved. Of all the joints, the hips, shoulders and knees are most markedly affected. The astragali and tarsal bones are slightly involved.

FIG. 6. Case II. Anteroposterior roentgenogram of the pelvis.

No other skeletal abnormalities were discovered in the course of a rather complete survey.

CASE III. V.R., female, aged thirty-eight. This patient is the mother of the females described in Cases I and II. Her delivery and neonatal development were not remarkable. At the age of sixteen months, it was first noted that she had difficulty in walking. The original abnormality of gait progressed slowly and was gradually supplemented by pain localized to the hip joints. The severity of pain increased until at the age of eighteen years she sought medical help. After a period of hospitalization she was told that she had a "flat hip joint and that her lower vertebrae were flat and fused."

Physical Examination. Height, 60 inches; weight, 100 pounds. Examination revealed a gait similar to that of her two daughters but an otherwise well developed and nourished female of indicated height and weight. Physical findings consisted solely of marked limitation of motion about the hip, elbow, and shoulder joints. However, she carries on her normal

Fig. 7. Case III. Anteroposterior roentgenogram of the pelvis.

household routine fairly well and has sufficient motion about her shoulder joints to comb her own hair. Laboratory examinations were normal.

Roentgenological Examination. Fairly complete bone and joint studies were obtained. The most revealing films are reproduced in Figures 7, 8, and 9. In this patient we see completely fused and well ossified epiphyses. The femoral heads, humeral heads and distal tibial articulations are markedly deformed. The opposing member of the joint in each instance shows definite coadaptive malformation. Advanced degenerative osteoarthritic changes are apparent in the hips and shoulders. Other films show only minimal involvement of knees and wrists and no involvement of the elbows. The ankle joints show definite obliquity of the joint space with malformation of the opposing surfaces of the tibiae and tali.

In view of the rather unusual familial incidence in the above cases, an attempt to discover the presence of this disease in other members of the family was made. Only historical information could be obtained. This, however, appeared to be fairly reliable. A genealogical chart summarizing the available information is presented in Figure 10. In this family group, this disease entity appears to be distributed as a dominant trait.

Case IV. (Courtesy of Dr. Reed S. Clegg, Salt Lake City, Utah).

W.G., male, aged five years. This patient was first seen in the course of seeking orthopedic help at the age of twenty-one months. At that time the history was that of normal delivery and no abnormality of neonatal growth and development. He first walked at the age of thirteen months. Nine months prior to the first orthopedic consultation, the patient fell down the basement steps at his home. No obvious injury was suffered but since that time he has been noted to limp occasionally. Subsequent to

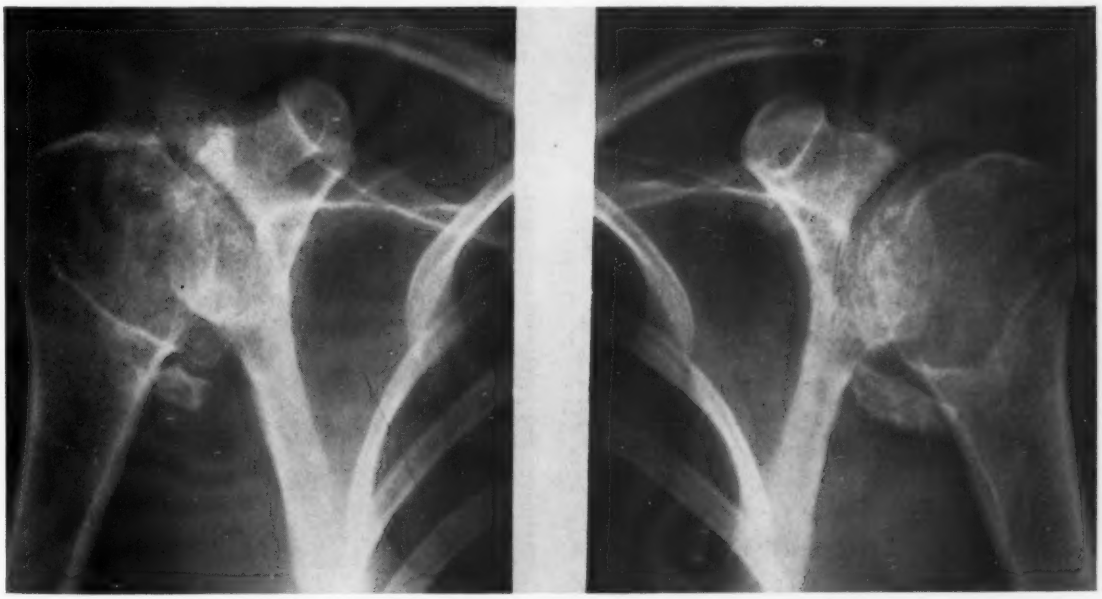

Fig. 8. Case III. Anteroposterior roentgenograms of the right and left shoulders.

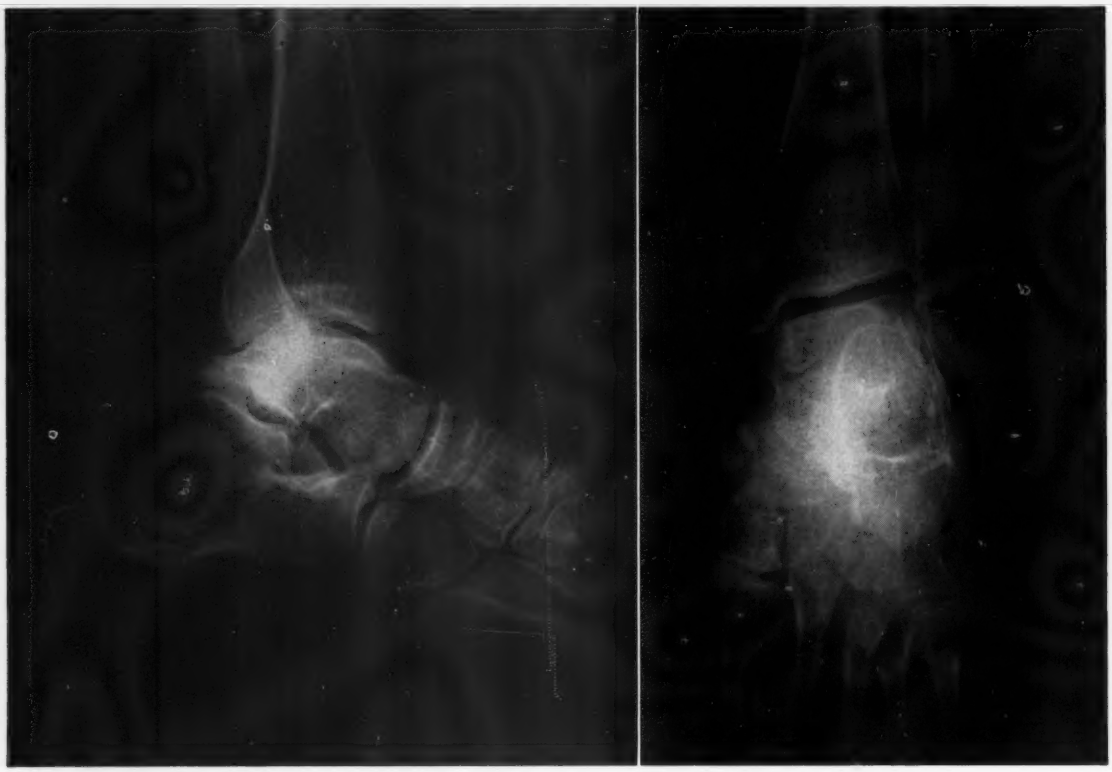

FIG. 9. Case III. Anteroposterior and lateral roentgenograms of the left ankle.

this first visit he has been regularly and carefully followed. He has continued to experience pain and difficulty in walking but his development has not been otherwise remarkable.

Physical Examination. Height, 42 inches; weight, 43 pounds. Orthopedic examination revealed an abnormal waddling gait but no other significant abnormality. The usual laboratory studies were not remarkable.

Roentgenological Examination. Studies obtained in the course of his last examination (aged five) are shown in Figures 11 and 12. The marked irregularity of the upper humeral epiphyses is clearly visualized. Ossification is proceeding from multiple primary centers. Even the ossification of the acetabula and iliac crests

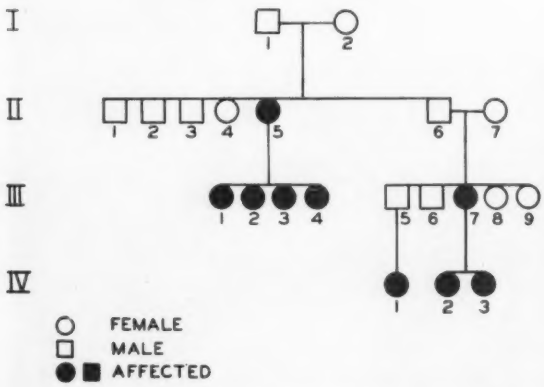

FIG. 10. Cases I, II and III. Genetic chart. Generation IV. No. 2. Case II (SR); No. 3. Case I (JR). Generation III. No. 7. Case III (VR). Mother of Cases I and II.

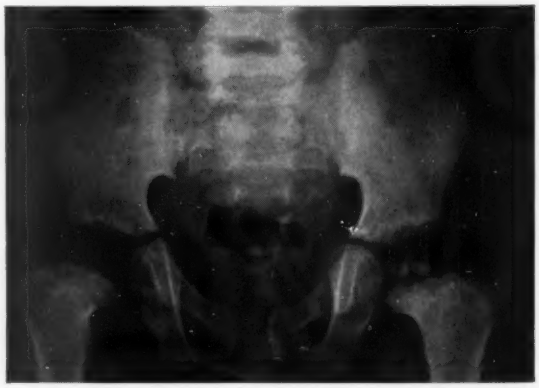

FIG. 11. Case IV. Anteroposterior roentgenogram of the pelvis.

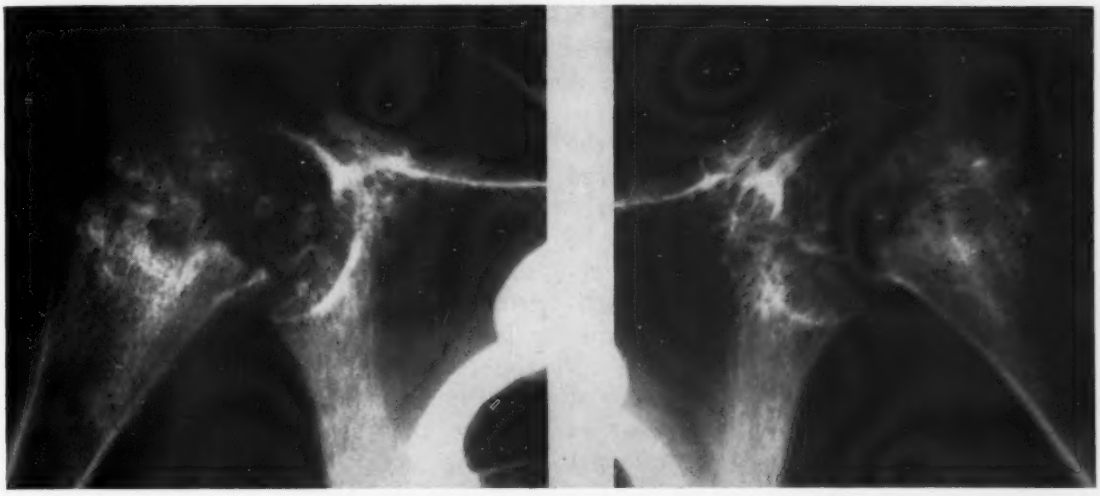

Fig. 12. Case IV. Anteroposterior roentgenograms of the right and left shoulders.

is somewhat more irregular than normally seen, but no deformity comparable to that seen in the femoral head is present. Other films show similar involvement of ankles, knees, elbows, and wrists, but the severity is much less than in the hips and shoulders. The digits are of normal size and configuration. The spine and skull and diaphyses of the long bones are not remarkable. The upper femoral metaphyses are obviously affected. The deformity here is much more marked than in any of the other cases.

DISCUSSION

There can be little doubt that these cases are closely related, if not completely similar. The three examples presenting in younger individuals, Cases I, II and IV are practically identical. It appears that a congenital developmental disorder has affected the epiphyseal structures of all the long bones. The signs and symptoms elicited are essentially what one would expect, namely, difficulty in walking, limitation of motion, and joint pain which varies in severity depending on the extent of the orthopedic abnormality. Although involvement sufficiently severe to produce gross joint deformity has been noted, none was seen in the present cases.

Comparatively speaking, the distribution of the defect in these examples is unusually widespread. While most of the major epiphyses have been recorded as showing evidence of the disease, involvement is generally restricted to one or two of these. Bilateral involvement of hips, shoulders or knees is the distribution most commonly noted. The wrists, elbows and small bones of the hands and feet are less frequently involved.

The accompanying figures exemplify the important roentgenological features of the disease. The epiphyses show the major involvement. Slight metaphyseal and no diaphyseal abnormalities are present. The architecture and density of the diaphyses are within normal limits. Involvement is bilateral and symmetrical. Contour of the epiphyses is abnormal. They are irregular in outline. While their general configuration in some instances suggests moulding changes due to the stress of weight-bearing, their surface irregularity is obviously due to failure of development of certain segments. The ossification of the epiphyses is most unusual. Multiple, irregular centers of calcification are present in the younger patients, *i.e.*, before fusion of the epiphyses. The process does not proceed from a single central zone. It is of considerable interest to note that the acetabulum and glenoid are essentially normal in the three younger cases in this group. On the other hand, marked irregularity is clearly observable in the older patient. This appar-

ently has occurred in response to the irregularity of the opposing joint surfaces and subsequent degenerative changes. A significant degree of osteoarthritis is apparent. Despite this, it is clear that the malformed epiphyses have finally achieved essentially normal ossification with satisfactory density and trabeculation. On the basis of these observations, one might regard the basic disease process as self-limited with a tendency toward spontaneous arrest. However, resolution and reconstitution do not occur. Maintenance of function is probably largely dependent on the severity of the deformity. The relatively satisfactory function which has been achieved in the case of our older patient is in striking contrast to the irregularity and mottling of the epiphyses of her children. Furthermore, she has received no definitive therapy. Spontaneous improvement probably does not occur, but joint function is generally better than one would expect on the basis of the appearance of the epiphysis.

In respect to the type and distribution of the epiphyseal defect, the cases correspond to the classical descriptions of Jansen and Fairbank, namely, a congenital disorder characterized by variation in density and irregularity of outline of several of the major developing epiphyses.[4,7] With what other skeletal disorders could this syndrome be confused? Fairbank has considered the differential diagnosis at some length. His discussion is of such completeness that we shall do nothing here but list what he considers to be the differential possibilities. They are as follows: osteochondritis, cretinism, dysplasia epiphysalis punctata, osteochondrodystrophy (Morquio-Brailsford type), dyschondroplasia, aseptic necrosis and osteopoikilia. Pituitary gigantism is an unusual consideration. All can be fairly readily differentiated from dysplasia epiphysalis mutliplex. Fairbank regards some cases reported as examples of multiple osteochondritis as rightfully belonging in this group.[8,10]

The etiology of this disease is completely unknown. As in the present cases, there are no reported instances of association with disorders of metabolism, blood dyscrasias or lues. No significant sex predilection has been observed. Familial incidence has been recorded in approximately half of the reported cases. The sex linkage observed in the family group presented is distinctly unusual. None of these cases shows dwarfism, stubby digits or mental retardation which are described as typical findings of the total dysplasia epiphysalis multiplex syndrome. While our patients are small in stature, they certainly do not fall into the dwarf category. Under such circumstances one wonders whether or not these abnormalities should be considered as identifying features. The diagnosis probably rests primarily on the basis of the unusual epiphyseal defects and should not be confused by the inclusion of such inconstant criteria.

SUMMARY

Four cases of an unusual congenital disorder of skeletal development termed dysplasia epiphysalis multiplex have been presented. This disease process largely involves the developing epiphyses. These show ossification from multiple, discrete centers and gross irregularity of outline. Some segments of the epiphyses appear to be completely undeveloped. In other respects, the appearance of the cases definitely suggests a component of moulding and stress shaping which could also be attributed to defective ossification or abnormal cartilage development. The irregularity and deformity of epiphyseal outline seen in advanced disease states would, of course, be consequent upon both of these defects. In view of these observations, we would suggest that the basic defect be described as "an abnormality of epiphyseal ossification." The metaphyses are rarely involved.

Normal, satisfactory trabeculation is finally achieved after fusion of the epiphyses has occurred. In this sense the disease may be regarded as self-limited or, more properly, subject to spontaneous arrest.

However, the sequelae of irregular articulating surfaces and coadaptive changes are never corrected. Early degenerative osteoarthritic changes are to be expected. Their severity and rapidity of development will depend upon the degree of epiphyseal deformity, which may vary considerably. No definite etiology has been established. Hereditary influences are of apparent importance. No therapy of proved value is available.

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CLINICAL AND ROENTGEN MANIFESTATIONS OF TARSO-EPIPHYSEAL ACLASIS

REVIEW OF THE LITERATURE AND REPORT OF AN ADDITIONAL CASE

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INTRODUCTION

THE purpose of this communication is to review the literature and record an additional case of an unusual developmental disorder of ossification centers called "tarso-epiphyseal aclasis." This anomaly may affect any of the osseous structures of the lower limb including the tarsals and the bones of the foot. It is manifested by eccentric cartilaginous overgrowth with consequent asymmetrical enlargement of the involved ossification centers. Our patient is now eight years old and has been observed at frequent intervals from the time he was eight months of age.

REVIEW OF THE LITERATURE

The first report of this condition was made in 1926 by Mouchet and Belot[9] who published the case of an eight month old boy with involvement of the tibia and astragalus. They used the title "La tarsomegalie." Their patient was observed until he reached the age of thirty months. Trevor[11] in 1950 reported a series of 10 patients with this abnormality and demonstrated the characteristic distribution of the lesion. He suggested the name "tarso-epiphyseal aclasis" for this disease. In a survey of the British and American literature, we found a case reported by Geyman[4] in 1931. The reproductions of his roentgenograms show changes in the knee and ankle which are highly suggestive of this anomaly. However, the clinical findings and description of skeletal involvement elsewhere in the body do not coincide with the entity as described by Trevor. Consequently it is not included in this review although it may well represent an example of tarso-epiphyseal aclasis.

Nature and Distribution of the Lesion. The abnormal ossification centers show eccentric overgrowth as the chondromatous portion enlarges asymmetrically. Diffuse amorphous calcific stippling and/or ossification is scattered throughout the anomalous cartilaginous mass. An apparent characteristic of the lesion is restriction of the overgrowth to one-half of the affected epiphyses of the long bones. An additional characteristic feature is noted when more than one epiphysis is involved in which instance all of the epiphyseal centers affected show overgrowth of either the medial or lateral portions. Thus, one limb may show involvement of the medial femoral and tibial epiphyses at the knee and the medial malleolus at the ankle. None of the previously reported cases have varied from this, nor does ours. It is noteworthy that in the only case in which both lower limbs were involved,[11] the medial condyle of the femur on the left and the medial aspects of the "ankle and foot" on the right were affected. However, in the cases with involvement of the bones of the foot, this pattern is not invariable. This is discussed in greater detail below.

Table 1 indicates the distribution of the involved epiphyses in the 10 cases including our own in which accurate information is available. The bones most frequently affected were the ossification centers of the distal femur, the astragalus, the distal tibia

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TABLE I

DISTRIBUTION OF OSSEOUS INVOLVEMENT IN TEN CASES

(Two cases in Trevor's series are omitted because the exact localization was not stated)

Ossification Center Involved	Side Affected and Locus of Involvement								Combined Total
	Right leg				Left leg				
	Medial aspect	Lateral aspect	Entire center	Total	Medial aspect	Lateral aspect	Entire center	Total	
Femur									
Proximal					1			1	1
Distal	1	1		2	5	1		6	8
Tibia									
Proximal					3	1		4	4
Distal					5			5	5
Fibula									
Proximal									
Distal		1		1		1		1	2
Astragalus		2	1	3	2	1	2	5	8
Scaphoid					2			2	2
Calcaneus		1		1					1
Cuneiform									
Internal					1		1	2	2
Middle					(1?)			(1?)	(1?)
First metatarsal							1	1	1
Phalanges (first toe)									
Proximal							1	1	1
Distal						1		1	1
Total	1	5	1	7	19 and 1?	5	5	29 and 1?	36 and 1?

and the proximal tibia. The patients exhibit no evidence of disease or associated anomaly other than the osseous abnormality. Case 2 had a cataract of the right eye. In all the other patients the deformities are muscular and/or osseous and are apparently the result of the underlying bony and cartilaginous overgrowth and consequent faulty joint mechanics. Growth and development, both physical and mental, are unimpaired. It is a noteworthy fact that of the 12 cases of this disease, 11 occurred in males.

Clinical Manifestations. As indicated in Table II, the parents are first made aware of the abnormality by mechanical interference with joint function and/or the presence of a mass. There is no pain, the patients appearing healthy and normal in every other respect. In the case seen at our clinic, the boy is active, of normal weight and his physical and mental development has not been remarkable with the exception of discrepancies in lengths of the legs and circumferences of the thigh and calf on the two sides. It should be noted that, in con-

trast to our case, in none of those reported by Trevor was there elongation of the affected limb.

Roentgenographic Findings. Initially, there is amorphous calcific stippling in the asymmetrically enlarged portions of the affected ossification centers. As the child grows older, the calcification becomes more extensive and irregular ossification takes place. When more than one epiphysis is involved, the changes are limited to either the medial or lateral aspects of the limb; *e.g.*, the lateral femoral and tibial condyles and the lateral malleolus at the ankle. The bony enlargement may extend anteriorly or posteriorly. When the bones of the foot are involved, the asymmetrical pattern of the changes is less pronounced.

TABLE II

CLINICAL AND ROENTGENOGRAPHIC MANIFESTATIONS IN 12 CASES

Case	Sex	Age*	Side Affected	Presenting Manifestations	Ossification Centers Involved	Clinical Observations at Initial Examination	Operative Intervention
1 Mouchet and Belot	Male	8 mo.	Left	Left talus valgus and prominence of medial malleolus	Medial malleolus, astragalus	Valgus of foot; limitation of motion at ankle, atrophy of thigh and calf muscles	
2 Trevor	Male	10 yr.	Right	"Swelling" of right ankle for 2 years	Lateral malleolus, lateral astragalus, calcaneus	Bony enlargement of lateral malleolus and astragalus, varus deformity at ankle, foot could not be everted. Cataract of right eye	Excision of lateral malleolar mass†
3 Trevor	Male	4½ yr.	Left	"Painless swelling" of left knee for 6 weeks	Medial femoral condyle	Bony prominence at medial aspect of knee. Painless motion through full range	Excision of medial femoral condylar mass†
4 Trevor	Male	8 yr.	Right	Swelling of right knee, history of trauma	Lateral femoral condyle, lateral astragalus	Bony swelling at lateral aspect of knee and ankle. Motion normal at knee except for "excessive rotation of the extended joint." Painless motion at ankle, but restricted to 20 degrees	
5 Trevor	Male	4½ yr.	Left	Left genu valgum for 2½ years with gradual increase in deformity	Medial femoral condyle and tibial condyle, medial malleolus, medial aspect of astragalus	Enlarged medial femoral condyle and tibial condyle, marked valgus deformity at knee which was held flexed with limitation of motion. Extreme valgus of foot with great limitation of ankle movements	Excision of bony mass at knee with arthrodesis. Second operation: bony masses of astragalus and medial malleolus excised†
6 Trevor	Male	7 yr.	Left	Valgus of left knee and foot	Medial femoral condyle, medial tibial condyle, medial malleolus	Bony swelling medial and posterior to knee. Limited motion at knee and ankle. Bony mass at medial malleolus	
7 Trevor	Male	1½ yr.	Left	Walking badly, dragging left leg and falling frequently	Lateral femoral and tibial condyles, lateral malleolus, lateral aspect of astragalus	Lateral bowing of tibia, swelling at lateral aspect of knee; later, lateral curvature of tibia increased and motion at knee became restricted. Changes progressed no more and knee motion improved	Biopsy only†
8 Trevor	Male	3 yr.	Left	Valgus deformity of foot	Medial femoral capital epiphysis, medial femoral condyle, medial malleolus, medial aspect of astragalus, scaphoid, internal cuneiform	Enlarged medial malleolus, valgus deformity of foot	
9 Trevor	Female	9 mo.	Right	Inability to extend right knee fully, and deformity of right foot	Medial femoral condyle, astragalus	Severe genu valgum and limitation of extension at knee; valgus of foot, correctable manually	Supracondylar osteotomy to correct valgus
10 Trevor	Male	6 yr.	Right	Data not available	"Inner side of right ankle and foot	"No functional disability"	
11 Trevor	Male	4 yr.	Left Right	Data not available Data not available	Medial femoral condyle "Medial ankle and foot"	Data not available	
12 D'Angio, Ritvo and Ulin	Male	8 mo.	Left	"Swelling" over medial aspect of ankle	Medial femoral condyle, medial tibial condyle, medial malleolus, astragalus, scaphoid, internal cuneiform, first metatarsal, proximal and distal phalanges of great toe	Bony swelling over medial aspect of ankle with limitation of inversion of foot	

* Indicates age of patient when first observed medically.
† Histopathologic study of tissue obtained at operation in these 4 cases showed "normal hyaline cartilage with areas of ossification."

Thus, in our case, the astragalus, internal cuneiform, the first metatarsal and proximal phalanx of the first toe show diffuse involvement which is not restricted to the lateral or medial portions. The same was true of the astragalus in 2 of the 7 other patients in whom this bone was affected. There may also occur precocious appearance of the ossification centers in various tarsal bones. These may at the onset present several irregular nuclei but do not in every instance participate in the dysplastic process. An example of this is Case 1. The centers of the scaphoid and the internal and middle cuneiforms appeared prematurely but they did not share in the irregular cartilaginous overgrowth of the astragalus and tibial malleolus, at least during the period of observation.

CASE REPORT

Robert B. (OPD No. 816948). Date of birth, May 14, 1946.

The patient was brought to the Boston City Hospital on January 8, 1947, at the age of eight months, because of a "swelling" in the region of the medial aspect of the ankle noted by his parents for the first time during the previous month. The mother had no difficulty during this pregnancy. There was no history of maternal rubella. Delivery was by forceps but otherwise was not remarkable. Birth weight, 7 pounds, 5 ounces. The growth since birth had been uneventful with normal mental and physical development. Vitamin intake had been ade-

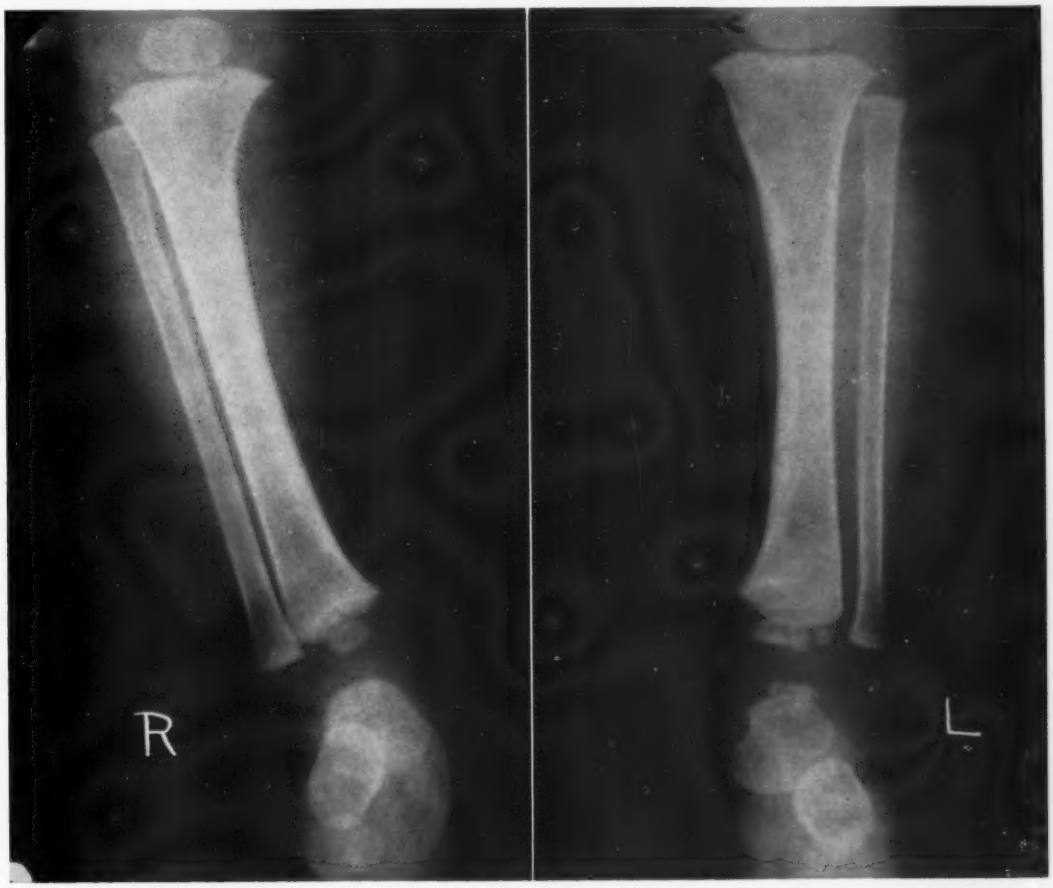

Fig. 1*A*. Lower legs, anteroposterior projection. Age eight months. The ossification centers of the lower end of the left tibia and the astragalus are enlarged asymmetrically. There is irregular calcification within the overgrown cartilaginous portions. The nucleus of the distal tibial epiphysis is larger than that on the right. The upper tibial epiphyses are symmetrical and appear normal in size and contour.

FIG. 1*B*. Lower legs, lateral projection. Age eight months. The enlarged cartilaginous portions of the left astragalus and tibia are irregularly calcified. There is anterior displacement of the left astragalus with relation to the tibia. The left tibia shows metaphyseal widening concomitant with the epiphyseal overgrowth; however, the distal portion of the diaphysis is well modelled and of normal size. Several small, irregular ossific nuclei indicate the precocious appearance of the internal cuneiform on the left.

quate. A serologic test (Hinton) was later reported as negative. The only sibling, a brother five years older than the patient, was normal. The parents presented no osseous abnormality. Physical examination showed a normal, well developed male infant except for a "bony swelling" over the medial aspect of the left ankle. There was no pain, tenderness, swelling or redness. The first roentgen study, made when the patient was eight months of age (Fig. 1*A* and 1*B*), showed asymmetry of the ankles with rounded densities in the regions of the astragalar ossification center and the distal tibial epiphysis. The nucleus for the latter was enlarged relative to the right. There were multiple scattered foci of ossification and calcification within the enlarged ossification centers which extended well beyond the usual limits of these structures. Also, there was anterior displacement of the astragalus on the tibia and precocious appearance of the internal cuneiform which ordinarily is not ossified until the third year of life or later.[5] The proximal tibial secondary center appeared normal.

His general growth and development proceeded uneventfully. He could stand alone at the age of thirteen months, and was able to walk unaided when eighteen months old, although with a slight limp. He could run and play actively when twenty-five months of age, at which time roentgen study (Fig. 2) showed muscle atrophy and osteoporosis of the entire left leg. Changes were then evident at the knee where, superimposed on the medial portions of the normal bony structures, there were irregular foci similar to those previously noted at the ankle. The tibia was sinuous and its axis was deviated lateralward. The diaphyseal length was 13.3 cm. compared to 13.0 cm. on the right. According to the standards of diaphyseal length

prepared by Maresh,[8] these measurements and others made subsequently place the patient near the tenth percentile. This is within the limits of normal and probably reflects hereditary factors since the parents and sibling are of medium build. In any event, there is no indication of significant retardation or impairment of growth of the long bones. The distal tibial epiphysis was greatly enlarged by comparison with its counterpart and the adjacent metaphysis showed concomitant widening. The astragalar abnormality showed progression with exuberant overgrowth and many scattered and confluent abnormal foci. The fibula was gracile. The fibular diaphyses measured 13.0 cm. on the left and 13.2 cm. on the right and there was an apparent relative delay in the development of the fibular epiphyses on the left. The proximal femur showed no epiphyseal change, but there was a suggestion of early valgus deformity.

Fig. 2. Lower legs, anteroposterior projection. Age two years, one month. On the left, the proliferation of bone and cartilage at the ankle has become more marked. There is gross distortion of the distal tibial epiphysis and of the astragalus. Early similar changes affecting the tibial and femoral epiphyses are present at the knee. The distal tibial metaphysis is widened slightly, but modelling is not impaired. There is genu valgum deformity and a sinuous curve of the left tibia. The bony structures on the left are osteoporotic, the fibula is slender and delicate in contour, and the musculature of the left lower thigh and calf is underdeveloped.

Fig. 3*A*. Lower legs, anteroposterior views. Age four years, ten months. The bony and cartilaginous overgrowths have progressed, particularly in the region of the knee where there are well defined bony masses with peripheral corticalis and central trabecular pattern. The overgrowths are in relation to the medial aspects of the left lower femoral and upper tibial epiphyses. The extensive involvement of the lower tibial epiphysis medially has resulted in definite enlargement of the internal malleolus. The deformed astragalus is approximately twice normal size. The left genu valgum, fibular and muscular underdevelopment, and osteoporosis are present as previously.

As the patient continued to develop, the inequality in leg lengths became more pronounced. It is of interest that the elongation on the left is the result of diaphyseal lengthening as well as the massive enlargement of the several ossification centers involved and the slight valgus at the hip. At the age of four years and ten months (Fig. 3*A* and 3*B*) the left lower leg roentgenographically was 26 mm. longer than the right; 11 mm. of the elongation was diaphyseal. Fibular diaphyseal lengths were 18.4

FIG. 3*B*. Lower legs, lateral views. Age four years, ten months. The ossific nucleus of the left patella is larger than on the right but the cartilaginous ossification centers show no discrepancy in size. The bony masses at the knee extend posteriorly into the popliteal space. The left astragalus shows massive overgrowth and gross distortion of outline. The adjacent articular surfaces of the os calcis are unaffected.

cm. on the right and 18.2 cm. on the left. There was a single ossific nucleus, measuring 1.2 by 0.5 cm., for the left patella. This was larger than the combined area (0.8 by 0.3 cm.) of the double nucleus present on the right but this amount of asymmetry is not uncommon in the patella during normal development. Moreover, the entire cartilaginous ossification center of the left patella has shown no evidence of disease during the period of observation. Other measurements at this time are shown in Table III.

As the child grew, all the bones along the medial aspect of the foot became involved until typical changes became evident in the astragalus, scaphoid, internal cuneiform, first metatarsal, and both phalanges of the great toe (Fig. 4 and 5). Possibly the middle cuneiform is also affected. The terminal phalanx shows, along its lateral margin, a bony crescent extending lateral to the interphalangeal joint. A slender fibula, narrowed metatarsal shafts, extreme muscle atrophy, and valgus deformity of the femoral neck on the left are apparently secondary to poor mechanics at the knee and ankle. Roentgen examination of the entire skeletal system shows sacralization of the transverse process of the fifth lumbar vertebra on the right and spina bifida occulta of the fifth lumbar and first sacral segments. Neither of these changes appears relevant to the disorder under discussion.

Clinical examination at the age of eight years revealed the following findings: At the knee, there was no gross deformity but there was a palpable, nontender, bony mass at the posterior medial aspect of the joint. Flexion was restricted to 90 degrees, extension was complete to 180 degrees. At the ankle there was marked enlargement and prominence of the medial malleolus, forward displacement of the foot on the tibia, and slight valgus position of the foot with prominence of the scaphoid on the medial aspect. The ankle was practically fixed at a right angle with only a few degrees of motion. The weight-bearing position of the foot was good, and the child walked with a barely detectable limp in spite of the stiffness of joints.

Treatment has been conservative with the use of lifts and wedges as indicated. Non-weight-bearing splints and regimens could not be utilized since the child is so active. He has been able to engage in all activities of his age group except for the inability to pedal a bicycle because of knee limitation.

DIFFERENTIAL DIAGNOSIS

Chondrodystrophia Calcificans Congenita (Stippled Epiphyses). In this condition there are scattered foci of calcification distributed throughout affected ossification centers. The epiphyses may be enlarged but are symmetrical and regular in outline. In tarso-epiphyseal aclasis, there is eccentric chondromatous overgrowth, the abnormal calcific foci being limited to the enlarged portions of the bone. In many cases of stippled epiphyses the diaphyses are short and thick with consequent dwarfing; there are flexion contractures at the affected joints often with para-articular fibrosis and calcification; there is mental dullness; and bilateral congenital cataracts are common. None of these are present in

TABLE III

COMPARATIVE OSSEOUS MEASUREMENTS AT THE AGE OF 4 YEARS AND 10 MONTHS

	Left	Right
Distal femoral epiphysis	5.2×2.3×3.0 cm.	5.7×2.3×3.0 cm.
Associated bony mass	2.2×3.7×2.0 cm.	
Proximal tibial epiphysis	3.2×4.1×1.6 cm.	2.9×3.8×1.6 cm.
Associated bony mass	3.2×3.5×2.2 cm.	
Distal tibial metaphysis	3.4×3.2 cm.	3.1×3.0 cm.
Distal tibial epiphysis	3.4×2.7×4.1 cm.	2.8×1.0×2.9 cm.
Astragalus	5.4×3.2×4.2 cm.	3.9×2.5×2.6 cm.
Calcaneus	4.7×3.2 cm.	4.4×3.2 cm.
Scaphoid	2.3×2.2 cm.	1.8×0.8 cm.
Internal cuneiform	1.2×1.7 cm.	1.0×1.1 cm.
Cuboid	2.0×2.1 cm.	1.9×2.0 cm.
First metatarsal—length	4.1 cm.	4.1 cm.
Minimum width	0.9 cm.	1.0 cm.
Second metatarsal—length	4.8 cm.	4.8 cm.
Minimum width	0.4 cm.	0.6 cm.
Proximal phalanx, great toe—length	2.1 cm.	2.1 cm.
Minimum width	0.4 cm.	0.6 cm.
Distal phalanx, great toe—length	1.7 cm.	1.8 cm.
Minimum width	0.8 cm.	0.7 cm.
Associated bony mass	0.7×0.2 cm.	

tarso-epiphyseal aclasis, and in the patient described by us there is diaphyseal lengthening rather than shortening. Common to both diseases is the occasional premature appearance of tarsal ossification centers, whether or not involved in the dysplastic process.

Geyman's case[4] is of interest in this connection since his reproductions show an appearance at the knee very suggestive of tarso-epiphyseal aclasis. However, changes in other bones are not incompatible with stippled epiphyses. Geyman's case may point towards a relationship between these conditions.

Chondrodystrophy (*Achondroplasia*). In this generalized condition, all the tubular bones are short with broad metaphyses. In the hyperplastic form they are often tufted and irregular. Particularly in this group the ossification centers may show spotty calcification, but there is no overgrowth of the center or other changes resembling the eccentric localized lesion in tarso-epiphyseal aclasis.

Dyschondroplasia (*Ollier's Disease*). Ollier's disease affects the metaphyses of the long bones and is characterized by columns and islands of proliferating cartilage, some of which undergo extensive calcification. In severe cases, the distortion is so great that the changes at the epiphyseal plate produce an appearance which might be confused with advanced tarso-

epiphyseal aclasis. However, in dyschondroplasia, there is predominantly metaphyseal involvement with secondary shortening of the diaphysis. While the epiphysis may undergo deformation conforming to the metaphyseal contour, it does not share in the chondromatous overgrowth and calcification which occur in the adjoining shaft.

Dysplasia Epiphysialis Multiplex. In dysplasia epiphysialis multiplex, the ossification centers appear late and develop slowly. They may be enlarged and irregular and present scattered calcific foci throughout their confines. The patients usually are short in stature because of the stunting of the diaphyses and may complain of pain and stiffness in the affected joints. Differentiation is established by the limited, asymmetric changes in tarso-epiphyseal aclasis and the absence of pain, diaphyseal shortening or delayed skeletal maturation. On the contrary, tarsal ossification often is premature.

Cretinism. Many hypothyroid infants show an aberration of skeletal development termed "cretinoid epiphyseal dysgenesis." This is characterized by irregularly ossified epiphyses, most commonly those of the proximal femur and humerus. While the

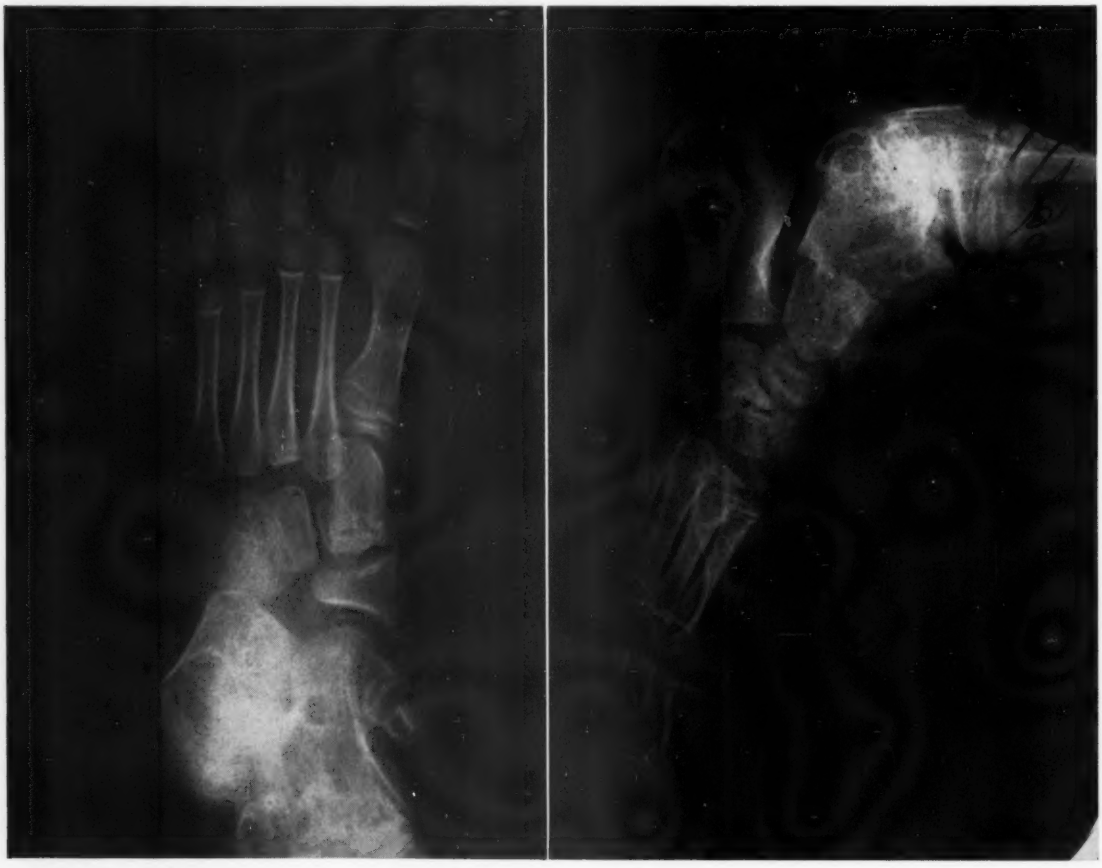

FIG. 4. Left ankle, posteroanterior and lateral views. Age five years, eleven months. All of the ossification centers along the medial aspect of the foot, including the terminal phalanx of the great toe, are affected. They are, however, at differing stages in the dysplastic process. The earliest manifestation is at the epiphysis of the proximal phalanx of the great toe where there is only scattered calcification beyond the normal confines of the ossification center. The first metatarsal shows along its dorsomedial aspect the next stage of the disease with a small bony outgrowth which is continuous with the ossific nucleus. Late, extensive enlargement is present in the internal cuneiform, the scaphoid, and the astragalus. The terminal phalanx of the great toe presents a crescentic bony mass along the lateral margin of the interphalangeal joint. There is narrowing of the middle thirds of the metatarsals.

Fig. 5. Left foot, anteroposterior and lateral views. Age seven years, one month. There is now evidence of enlargement of the cartilaginous portion of the epiphysis of the proximal phalanx of the first toe and "stippling" as previously. The involvement of the epiphysis of the first metatarsal is more pronounced, and the abnormal bony mass along the distal medial margin of the astragalus is larger than it was fourteen months before (compare with Fig. 4).

appearance may be similar to that in the early stages of tarso-epiphyseal aclasis, the affected epiphyses in the cretin tend to be smaller than normal in size, irregular in outline, and without eccentric osteochondromatous masses. The characteristic facies of the cretin, the retarded mental and physical development, and dwarfism establish the differentiation between tarso-epiphyseal aclasis and cretinism.

Aseptic Necrosis. Primary aseptic necrosis of the astragalus (Mouchet), tarsal scaphoid (Köhler), medial cuneiform (Buschke) and medial tibial condyle (Blount) affect some of the bones frequently involved in tarso-epiphyseal aclasis. Points of similarity in the two diseases include irregular ossification and fragmentation of the affected nuclei. However, painless eccentric epiphyseal overgrowth and calcification, the essential features of tarso-epiphyseal aclasis, are absent in osteochondritis.

Calcified Hematoma. Amorphous calcification may take place in hematomas. When located near a joint, it may be confused with a disturbance of the epiphysis. The calcification is periarticular and not in cartilage continuous with the ossification center as occurs in tarso-epiphyseal aclasis.

DISCUSSION

The etiology of tarso-epiphyseal aclasis is unknown. Laboratory investigations, including serologic studies, reveal normal

findings, there is no demonstrable relation to trauma or infection, and no evidence that the changes are neoplastic in character. It is evidently developmental in origin, as suggested by Trevor, who postulates a disruption of the normal cellular activity of the cartilaginous ossification center. This normally follows an orderly sequence of cell proliferation, maturation, senescence, and disintegration. According to Trevor's thesis, cells from one portion of the "mitotic annulus" (Harris[6]) continue to multiply unduly without following the usual processes of maturation and eventual cell death. The term "aclasis" used in the title derives from this failure of cellular breakdown. Because of the striking constancy of the pattern of osseous involvement, we have attempted to find an explanation on the basis of the embryology of the lower extremity. There is, however, relatively little specific information with regard to the early development of the limb bud. The most revealing studies are those of Steiner[10] in the field of comparative embryology. His investigations, together with the homologies of the tarsal and carpal bones,[1] help to explain the diffuse involvement of the ossification centers of the bones of the foot which occurred in our case. No definite correlation is evident between the embryology of the tibia and fibula and the pattern of the lesion in these bones. Trevor suggests a possible relationship between the hemi-epiphyseal distribution of the dysplasia and the blood supply of the epiphysis. According to Harris,[6] nutrient vessels course along the joint capsule, enter the nucleus and supply the adjacent portion of the epiphysis, terminating in an anastomotic network. In the presence of such a relationship in the local blood supply, it becomes possible to ascribe some of the findings to the effects of local hypervascularity. This could account for the premature appearance of ossification centers which often occurs in this disease, whether involved in the dysplastic process or not. It also would aid in explaining the diaphyseal elongation which occurred in our patient. While there is no overt evidence of local hyperemia or of an imbalance of the autonomic nerve supply, the distribution of the lesion corresponds well with the segmental innervation of the ossification centers and consequently of the blood supply.[3,7] For example, in our case, a fasciculus of a nerve root, the fifth lumbar, supplies the involved portions of all the affected ossification centers. The correlation is best demonstrated at the knee where the osteochondromatous proliferations present medially project only posteriorly, the anterior borders of the ossification centers remaining normal. It is to the posterior portion of the tibial and femoral epiphyses that fibers of the fifth lumbar nerve root are distributed. It also supplies the posterior aspect of the medial malleolus, and it is primarily this portion of the lower tibial epiphysis which developed the abnormality. A similar relationship can be established in the bones of the foot.

The process is a progressive phenomenon, at least to the beginning of puberty. Different nuclei become affected successively. In the patient described by us, the disease manifested itself at various times as the child grew older and its progress could be traced in the ossification centers of the proximal tibia, the internal cuneiform, and the great toe metatarsal and its phalanges.

In addition to the remarkably similar clinical and roentgenographic manifestations which occur in the 12 cases included in this review, our patient presents certain additional features. (1) There is lengthening of the affected leg, partly because of the massive enlargement of ossification centers and coxa valga, and partly because of diaphyseal elongation. The case reported by Mouchet and Belot[9] developed 2 mm. of increased length in the first and second metatarsals on the affected side, although these bones were not involved. This is the only other instance of osseous elongation in any of the previously reported cases and it is questionable whether inequality of this extent is valid. (2) More ossification centers are involved than in any of the other cases. All nine nuclei along the medial

aspect of the leg from the knee to the great toe show characteristic changes and there is also probable early involvement of a tenth nucleus, that of the middle cuneiform. (3) The involvement of the epiphysis of the terminal phalanx of the great toe along its lateral margin seems to deviate from the predominantly medial distribution of the asymmetrical lesions in the other epiphyses. The fifth lumbar nerve root supplies the entire epiphyses of the first metatarsal and the great toe phalanges. Therefore, should the involvement, as suggested above, be related to the segmental innervation, the aberration thus could affect any or all portions of these ossification centers.

There has been no indication of a neoplastic tendency in the hypertrophied chondromatous tissue. The available biopsy reports show normal hyaline cartilage with foci of calcification and ossification with no evidences of rapid growth, metaplasia or neoplasm formation. Nonetheless, cartilage proliferating abnormally should always be viewed with suspicion and we plan to continue to observe our patient with this potentiality in mind.

TREATMENT

In our case, as in many of those previously reported, conservative management with braces and special shoes suffices. The cartilaginous proliferation continues during the early years. With the advent of puberty, the progression of the disease may be expected to cease. This is implied by the eventual arrest of the condition in one of the patients in Trevor's series who was observed for fifteen years and therefore presumably beyond pubescence. If the patient can be maintained through the years before puberty with conservative measures, it appears that there is favorable prospect for eventual arrest of the developing bony masses. Should surgery be necessary, it may comprise (1) local excision of the cartilaginous mass; (2) excision of the abnormal growths with arthrodesis; and (3) osteotomy and other plastic procedures to correct valgus and varus deformities.

SUMMARY

The clinical and roentgenographic features of tarso-epiphyseal aclasis are presented, the literature is reviewed and an additional case is reported.

Tarso-epiphyseal aclasis is a congenital error in development which may affect any of the ossification centers of the lower limb. It is manifested by painless eccentric overgrowth of involved nuclei, and may lead to mechanical interference with joint function. Otherwise there are no associated anomalies. The condition is characterized by restriction of the involvement to either the medial or lateral aspects of the epiphyses of the long bones. In the foot, the dysplastic pattern is variable.

The etiology is unknown. The possible relationship to epiphyseal blood supply was first mentioned by Trevor. Certain characteristics of the disease suggest the presence of local hyperemia. A possible relationship to vasomotor innervation is indicated because of the close correlation between segmental nerve supply by a single lumbar spinal root and the characteristic pattern in certain instances.

The case reported by us differs from those previously described in the literature in three respects: (a) more ossification centers are involved; (b) there is lengthening of the affected limb which is caused in part by diaphyseal elongation; (b) the terminal phalanx of the great toe is involved along the lateral aspect. This differs from the pattern in the leg in which involvement is predominantly medial in distribution. An explanation is suggested for this divergence.

The differential diagnosis and treatment are presented.

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LEIOMYOSARCOMA OF THE SMALL INTESTINE*

A REPORT OF THREE CASES AND REVIEW OF THE LITERATURE

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LEIOMYOSARCOMA is an uncommon tumor in the intestinal tract. The relative difficulty encountered in the roentgenographic diagnosis is determined by the size and position of the tumor which may be intraluminal, intramural, or extraluminal. The clinical and diagnostic features of two such lesions occurring in the small intestine are noted in the following report.

HISTORICAL REVIEW

The first reported leiomyosarcoma of the small intestine was by von Salis[14] in 1920. In 1939, Foshee[5] reviewed 4 additional cases and presented 1 of his own. Others have since been reported by Harrington and Ganshorn,[8] Henning and Garland[9] and McCullough.[12] Heymann and Clark[10] noted 15 cases of duodenal leiomyosarcoma in the literature and reported 1 of their own. Geschickter[7] reported 20 malignant tumors of the small intestine, 1 of which was a myosarcoma. Dundon[3] reports 18 cases of malignant tumors of the small intestine, 12 of which were carcinoma, 2 lymphosarcoma, 2 malignant carcinoid, 1 fibrosarcoma and 1 leiomyosarcoma. In a review of 92 malignant tumors of the small intestine seen at the Mayo Clinic from 1907 to 1939, 87 per cent were adenocarcinoma and 10.8 per cent were leiomyosarcoma.[13] The location of the latter was as follows: duodenum, 2; jejunum, 2; ileum, 1; Meckel's diverticulum, 1. In 1, the location was not specified. Additional information as to distribution is contained in a review of the literature from 1932 to 1941 by Frank *et al.*,[6] which discusses 102 instances of sarcoma of the small intestine and includes 22 leiomyosarcomas and 1 myosarcoma. The sites of the latter were duodenum, 7; jejunum, 7; ileum, 7; jejunum-ileum, 1; and Meckel's diverticulum, 1.

ANATOMIC AND PATHOLOGIC CONSIDERATIONS

The leiomyosarcoma is a tumor which originates in smooth muscle and may occur wherever this type of tissue is found. It may be either single or multiple. When growth is primarily into the submucosa, it may present as an intraluminal polypoid lesion having either a pedunculated or a sessile base. When growth is primarily towards the serosa, it extends away from the lumen of the viscus and presents as an extraluminal mass[8] which tends to displace the surrounding organs and to become adherent through the formation of adhesions.[4] Central necrosis, ulceration, fistula and sinus tract formation, perforation and abscess formation may occur. McCullough[12] reports a case in which the specimen contained many branching sinus tracts.

Grossly, the tumors are firm and elastic and vary in size from a small nodule a few millimeters in diameter to large irregular masses 15 cm. or more in diameter. The capsule may be smooth and complete. The cut surface reveals a homogeneous mass which may contain areas of necrosis, hemorrhage, ulceration, fistula and sinus tract formation and even calcification. The serosa may be smooth or reveal evidence of inflammation or fibrosis forming adhesions to surrounding organs. The entire tumor or portion of it may be incorporated in an abscess.

The microscopic study reveals the tumor to be composed of irregular whorls of smooth muscle tissue and varying amounts of connective tissues. The smooth muscle

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cells appear bizarre, show considerable variation in size and are usually shorter than normal. The nuclei vary in character and staining reaction and demonstrate evidence of abnormal mitoses.

CLINICAL FEATURES

The presenting complaints usually are those of: (1) hemorrhage, manifested by melena, weakness, anemia and fatigue; (2) abdominal pain and distress of varying degree; (3) the presence of a palpable mass.[12] Jaundice may occur if the tumor is located at the ampulla of Vater or produces compression of the common bile duct.[10,16] Nausea and vomiting are infrequent. Loss of weight may be pronounced. Intestinal obstruction may be produced by the increasing intraluminal growth of the tumor. The symptoms of obstruction may be recurrent in character. Temporary improvement has been ascribed to sloughing of the tumor mass. It is possible that intussusception[10] may develop and produce intermittent episodes of cramp-like abdominal pain and bleeding but this has not been recorded in the literature reviewed.

ROENTGEN FEATURES

The roentgen findings in leiomyosarcoma of the small intestine depend upon the location of the tumor in the wall. The intraluminal type may present: (1) a filling defect with sharp, smooth, rounded borders; (2) unaltered peristalsis except at the base of the tumor; (3) distortion of the overlying and adjacent mucosa, and (4) superficial ulceration on the dome of the tumor. These features are also common to the intraluminal leiomyoma and differentiation between these two is dependent upon the histopathologic examination. As the tumor grows and larger areas of ulceration and necrosis develop, the margins become irregular and the lumen of the small intestine may be narrowed.

The extraluminal leiomyosarcoma produces: (1) an extrinsic mass and pressure defects with varying degrees of narrowing of the lumen; (2) distortion and actual destruction of the adjacent mucosal folds; (3) ulceration; (4) interruption of the normal peristalsis, and (5) fistula, sinus tract and abscess formation.

Superficial ulceration on the dome of the tumor has been demonstrated in the gross specimens and on the roentgenograms of the intraluminal leiomyosarcoma. This finding in not pathognomonic of these tumors but is present with a high degree of frequency when they are identified. Deep ulceration and fistula formation into the extraluminal portion of the tumor have been described in the resected specimens of leiomyoma,[11] the leiomyosarcoma as well as in lymphomas and in primary melanoma.*

Schindler and his associates,[15] in 1946, reported 4 patients with leiomyosarcoma of the stomach in whom roentgen and gastroscopic studies had been performed. The roentgenograms in 1 of his cases revealed "two niche-like projections on the postero-medial side of the stomach at the junction of the proximal and middle thirds." The resected specimen revealed an ucler 1.5 by 0.6 cm. in size. This author described the roentgen syndrome as show a filling defect with a central niche and fistulas which "should be considered as highly suggestive of leiomyosarcoma."

Fistula formation is also mentioned by Foshee and McBride[5] as being present in 4 of the cases he studied, and abscess formation had occurred in 1 instance. McCullough[12] suggested the sequence of ulceration-sinus tract formation and perforation as being important in these tumors. He reported 2 additional cases in which fistulas were present. Deep ulceration and fistula formation was described by Collins *et al.*[2] in a leiomyosarcoma of the jejunum. The roentgenogram was described as demonstrating "a small diverticulum of the jejunum about six inches from the ligament of Treitz." The area of ulceration measured 1.4 by 2.3 cm. in size. Jones and

* From the files of the Department of Pathology, Bowman Gray School of Medicine of Wake Forest College. Courtesy of R P. Morehead, M.D.

Brubaker[11] reported a leiomyoma of the ileum in which the tumor was completely encapsulated but contained a fistula. Skinner and Walters[17] described a leiomyosarcoma in a Meckel's diverticulum in which there was fistula formation and perforation.

The following case reports are illustrative of many of the described diagnostic features.

REPORT OF CASES

CASE 1. *Intraluminal Leiomyosarcoma of the Duodenum Presenting with Ulceration and Hemorrhage.*

The patient was a white male, aged thirty-seven, who was first admitted to the hospital on Feb. 14, 1947, with a chief complaint of an insidious onset of weakness and blood in the stool. This was first noted in 1945 and he had experienced periodic episodes of bleeding and weakness since that time. He had not noticed pain and, although he was placed on an ulcer regimen, the bleeding persisted.

FIG. 1*A*. Case 1.* Examination of the stomach and duodenum in February, 1947, shows no detectable evidence of a lesion in the descending portion of the duodenum.

FIG. 1*B*. Case 1. Examination in August, 1949, reveals an intrinsic filling defect in the descending duodenum with flattening of the muscosal folds.

At the time of admission, the hemoglobin was 73 per cent and the red blood cell count was 3.84 million. Roentgenographic studies of the esophagus, stomach and duodenum revealed a small inconstant hiatus hernia. He was discharged with a diagnosis of gastrointestinal hemorrhage, cause undetermined.

The patient was readmitted on March 17, 1947, because of repeated episodes of tarry stools. The physical examination was negative but the laboratory studies revealed the hemoglobin to be 62 per cent and the red blood cell count to be 3.22 million. The roentgenographic studies of the upper gastrointestinal tract, including the small intestine, were negative. A string passed into the stomach revealed a

* This case is presented through the courtesy of Boyd G. King, M.D., and H. L. Friedell, M.D., University Hospitals, Cleveland Ohio.

FIG. 1*C*. Case I. Spot roentgenogram demonstrates the intraluminal defect, abnormal mucosal folds and the small superficial ulceration.

blood stain estimated to be at the level of the pylorus. Gastroscopic examination was negative but was thought not to exclude ulcer. He was again discharged on an ulcer regimen.

The third admission was on April 30, 1949 because of acute anemia due to blood loss. The roentgenographic studies of the upper gastrointestinal tract again failed to disclose the cause of the bleeding and he was discharged on May 4, 1949.

The fourth admission was on July 31, 1949, because of tarry stools of twenty-four hours' duration. The general physical examination was negative. The roentgenoscopic and roentgenographic studies revealed a filling defect in the descending duodenum measuring 5 cm. in diameter with moderate distortion and flattening of mucosal folds and a small accumulation of barium in the central portion which was thought to represent a small ulcer niche.

Surgical exploration revealed an umbilicated tumor under the mucosa of the posterior wall of the duodenum, immediately below the ampulla of Vater. There was a small superficial ulcer on the dome of the tumor. Several large soft lymph nodes were present behind the duodenum. The tumor shelled out easily leaving a small perforation on the posterior duodenal wall which was closed.

The microscopic examination revealed a nodular mass of elongated, pink-staining spindle cells just below the mucosa. The nuclei were large, oval and hyperchromatic. An occasional mitotic figure was seen. The diagnosis was leiomyosarcoma of the duodenum. The lymph node revealed hyperplasia.

Postoperative roentgenographic studies revealed a normal duodenum. The patient has been followed for four years and there has been no evidence of recurrence of the tumor or the gastrointestinal bleeding.

CASE II. *Extraluminal Leiomyosarcoma Manifested by Pain, Palpable Mass and Fistula Formation.*

The patient was a white male, aged fifty-five, who was admitted to the hospital complaining of episodes of vomiting and generalized post-

FIG. 2*A*. Case II. Small intestinal series demonstrates the lesion in the left lower quadrant. The barium-filled fistula is easily identified. The jejunum is narrowed and the mucosa is distorted.

prandial pain of four years' duration. Three months prior to admission, he experienced sharp, intermittent, stabbing, lower abdominal pain which radiated to the umbilicus and lasted for varying periods up to several hours. At this time, a slightly tender mass was palpated in the left lower quadrant. There had been a weight loss of 30 pounds in the three months prior to admission. A small amount of dark blood was noted with the stool on one occasion; otherwise, the stools had remained normal in frequency and character.

The positive physical findings were limited to the abdomen which was slightly protuberant. The liver was palpable 3 cm. below the right costal margin. An irregular, fixed, slightly tender mass about 8 by 6 cm. in size was palpated in the left lower quadrant, the superior margin of which was just below the umbilicus. The laboratory data revealed red blood cell count of 4.05 million and hemoglobin 11.5 gm.

The small intestinal roentgen series revealed no evidence of obstruction but there was slight hypermotility. The barium reached the cecum by the end of one hour and the column appeared fragmented. An area of narrowing of the lumen of the jejunum was identified in the left lower quadrant with measured 6 cm. in length and throughout which the mucosa was thickened, frayed and distorted. A pear-shaped fistula 6.0 by 2.5 cm. in size extended from the mid-portion of the constricted area. The margins of the fistula were irregular and serrated. One of the studies revealed barium within the lumen of the fistula. The adjacent loops were displaced by the tumor mass in the jejunum.

Fig. 2*B*. Case II. Spot roentgenogram demonstrates the fistula and the displacement of the adjacent small intestine by the tumor mass.

Operative Note. Surgical exploration of the abdomen revealed a tumor mass in the left lower abdomen about 30 inches from the ligament of Treitz. It was lobulated in character and, in one portion, almost completely encircled the bowel. It was firmly attached to the anterior parietal peritoneum at the level of the semilunar line of Douglas, to the superior aspect of the urinary bladder and to an adjacent loop of distal ileum. During the process of dissection, it was inadvertently incised, revealing a large area of central necrosis. A 20 cm. segment of jejunum and an 8 cm. segment of adherent ileum were resected. The jejunum was involved for a distance of 10 cm. and within the lumen, there was an area of ulceration.

Pathology. The gross specimen consisted of a 20 cm. segment of jejunum with an irregular, gray, friable, firm mass in the mesenteric portion measuring 10 by 8 by 5 cm. in size. A central area of necrosis 3.5 cm. in diameter was present in the mass of the tumor. An ulcer was present within the lumen of the jejunum which measured 2.0 by 1.0 by 2.0 cm. No communication was demonstrated between the ulcer and the central area of necrosis. The microscopic studies revealed the mass to be composed of cells which varied in size and staining reaction. They closely resembled smooth muscle cells in some areas and bizarre connective tissue cells in others. The sections of ileum revealed minimal infiltration with inflammatory cells.

Diagnosis. Leiomyosarcoma of the jejunum with extension into the mesentery.

Case III. *Extraluminal Leiomyosarcoma with Pain, Palpable Mass and Fistula Formation.*

The patient was a white male, aged sixty-six, who was admitted to the North Carolina

Fig. 3*A*. Case III. Examination of the small intestine reveals the tumor mass with the barium-filled fistula in the upper jejunum.

Baptist Hospital complaining of episodes of severe cramp-like generalized abdominal pain of one year's duration. There was no history of constipation, distention or vomiting, but he had experienced nausea and anorexia. The episodes were not related to meals. Two weeks prior to admission, he had a bout of abdominal pain which was followed by a shaking chill and fever. He had lost 8 pounds in weight in the five months prior to admission.

The physical examination revealed the temperature to be 101.6° F. (oral). There was a freely movable soft mass palpable in the left lower quadrant which was tender to palpation and measured 4 cm. in diameter. The rectal examination revealed enlargement of the prostate gland, which was smooth and firm. Proctoscopic examination revealed the presence of dark blood in the lower sigmoid colon.

The laboratory findings revealed the hemoglobin to be 10 gm. The red blood cell count was 3.3 million and the white blood cell count was 7,500. The urine contained 25–30 white blood cells per high power field. The stool gave a positive reaction for occult blood, but was negative for ova and parasites. Febrile agglutination tests revealed a positive agglutination for paratyphoid B in a 1:320 dilution and to proteus OX19 in a 1:160 dilution.

The patient had a febrile course in the hospital. On the ninth hospital day, he was given a glycerine and water enema in preparation for pyelography and afterwards he stated that he had passed a small, sharp object per rectum which he did not observe. Following this, he became afebrile.

Roentgen studies revealed an abnormality in the jejunum as demonstrated in Figure 3*A*. This was interpreted as being either a granulomatous mass or tumor. The patient was discharged to return in two weeks for re-examination.

He was readmitted to the hospital thirteen days later. The examination revealed little significant change in the lesion in the jejunum except for a slight reduction in the size of the fistula. There was a mass palpable in the left lower quadrant which was associated with the jejunum and contained an irregular fistulous mass 4 by 2 cm. in size. The provisional clinical diagnosis was leiomyosarcoma of the jejunum with ulceration and fistula formation.

Fig. 3*B*. Case III. Examination three weeks later reveals a slight reduction in the size of the fistula in the jejunal tumor.

Fig. 3*C*. Case III. Spot roentgenogram demonstrates the irregular margins of the fistula and the slight deformity of the jejunal lumen.

Operative Note. Surgical exploration of the abdomen revealed a hard loculated mass, predominantly in the mesenteric border of the jejunum approximately 12 inches from the ligament of Treitz. There were numerous peritoneal and omental adhesions. Several enlarged mesenteric lymph nodes were identified. There was no evidence of obstruction. Several small nodules were present in the liver which were thought to represent metastatic tumor. The jejunal tumor was resected and an end-to-end anastomosis was performed. The recovery was uneventful.

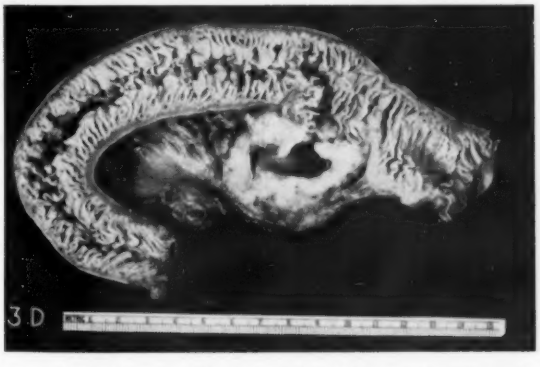

Fig. 3*D*. Case III. Photograph of the resected specimen in the sagittal plane demonstrates the actual relationshiop of the extraluminal leiomyosarcoma to the jejunum.

Pathology. Microscopic study reveals a tumor which arises within the muscular coasts of the jejunum. It consists of interlacing bundles of smooth muscle cells which are anaplastic, pleomorphic, basophilic and show a moderate number of mitotic figures. The cavity noted within the growth contains necrotic tissue and granulation tissue along with an exudate with a predominance of lymphocytes. Trichrome stains show the bundles of tumor cells to stain as smooth muscle cells. Reticulum stains are consistent with a smooth muscle tumor. Diagnosis: Leiomyosarcoma of the jejunum.

DISCUSSION

A study of the leiomyosarcoma of the small intestine suggests a definite growth pattern. The tumor originates from smooth muscle cells in the wall of the viscus. It may grow either into the lumen, within the wall of the viscus or extend subserosally into the peritoneal cavity. Being relatively avascular, it is not surprising that central necrosis, ulceration and fistula formation may occur as the tumor increases in size.

The roentgen method is useful in detecting these lesions when they are sufficiently large to produce deformity of the mucosa, a filling defect within the lumen of the hollow viscus or a mass lesion within the abdomen. Those tumors that are intraluminal or intramural may be identified while the mass is relatively small. The tumors which are predominantly extraluminal and extramural are usually identified only after they have reached considerable size or after they produce gross deformity or displacement of adjacent organs. This difference in growth pattern most likely is responsible for the relatively low incidence of detection of the extraluminal type (Chaffin[1] quotes 28 per cent negative roentgen examinations in this type of lesion in the stomach).

The extraluminal leiomyosarcoma presents roentgen and pathological features which differ from the interluminal type. In the former, the mass is usually larger before identification is possible, giving a greater probability of necrosis, ulceration and fistula formation. The gross descriptions of the specimens in the literature do

not permit accurate evaluation as to the frequency of occurrence of fistula formation in the extraluminal tumors but they do suggest that it occurs more commonly in this type of lesion.

The cases in this presentation support this contention. Since the roentgen method is better adapted to the identification of the intraluminal defects, it is possible that they are recognized prior to fistula formation or they may produce more profound clinical symptoms which demand surgical intervention.

The diagnosis of leiomyosarcoma is to be considered when fistula formation within a tumor mass is identified. This finding, however, is not specific for it may occur with leiomyosarcoma, leiomyoma, lymphosarcoma, melanoma, and inflammatory diseases of the small intestine. The fistulous tract observed in the case presented and those in the literature have had grossly irregular margins. This serves as a diagnostic aid in differentiating simple diverticulum, although it is possible that a diverticulum complicated by either inflammation or tumor could present a similar appearance.

SUMMARY

Three instances of leiomyosarcoma of the small intestine are reported. One occurred in the duodenum and was intraluminal in type, producing a filling defect within the lumen and exhibiting a small ulceration on the dome of the tumor mass. The gross appearance was that of a benign lesion, but the histopathologic findings were those of leiomyosarcoma.

The other two were the extraluminal type with the greater portion of the tumor extending away from the lumen of the small intestine, producing only slight narrowing of the lumen of the small intestine but containing a rather large fistula.

Ulceration, central necrosis, fistula and abscess formation have been shown to occur in leiomyosarcoma with surprising frequency. Although these findings are not pathognomonic of this lesion, the diagnosis of leiomyosarcoma must always be considered when they are present.

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ROENTGENOGRAPHIC OPACITY OF THE HEPATIC CIRCULATION*

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THE difficult access to organs deeply situated in the abdominal cavity, such as the pancreas, liver and spleen, has made direct roentgenological exploration difficult or impossible. In such cases the indirect signs given by the exploration of the adjacent viscus are the only roentgenological evidence on which to base the diagnosis.

Angiocardiography has made possible the exploration of a considerable portion of the vascular system, and it seemed feasible to apply the same principle to the study of the hepatic circulation.

The object of this paper is to present the results obtained by the injection of an opaque substance and the roentgenological exploration of the suprahepatic vascular system.

HISTORY

The roentgenological exploration of the liver has been tried repeatedly by different students following diverse techniques. Oka[10] in Tokio, and Radt[12] in Berlin in 1928 experimented with a preparation of thorium injected intravenously. Afterwards this procedure, hepatosplenography, was tried by a score of investigators, among whom we may cite Yater,[17,18,19,20] Cooke,[7] Whitaker,[16] and Tripoli,[15] who used a stabilized colloidal solution of thorium dioxide as a contrast substance.

When injected in the blood stream, this substance is fixed rapidly by the reticuloendothelial cells, and permits the roentgenological visualization of the spleen and liver as a homogeneous shadow, sometimes traversed by more opaque streaks: the main blood vessels.

In pathological cases the shadow assumes a mottled aspect because the fibrous tissue does not fix the thorium, thus leaving radiotransparent spaces.

The reactions produced by the use of thorium salts, according to the above mentioned authors, are slight or nonexistent and they are, it seems, innocuous to the liver cells. The substance may remain in the liver as long as ten years, and this fact prompted the Council on Chemistry and Pharmacology of the the American Medical Association to warn the medical profession against the use of radioactive substances that might afterwards produce unwanted effects.

The elimination of this substance is made partially through the lymphatic system, thus accounting for the shadows observed in the lymph glands.

In Mexico City, the technique of Oka and of Radt has been used with satisfactory results by Dr. Bassols. A. Cancino, in 1947, studied the arterial and portal circulation of the liver of the cadaver, injecting the vessels with lead protoxide. R. Flores López[8] studied the pancreatic arterial system injecting the same substance through the right gastro-epiploic or the splenic arteries.

In 1949, Olsson and Ekman[11] successfully performed hepatography in rats fed with a mixture of halogen substances and fatty acids which are absorbed in the intestines and resynthesized in the liver. They used 1 to 4 mg. of bromine in sesame oil. After twenty-four hours, the liver could be seen roentgenologically as a dense shadow.

In a conference at the Instituto de Cardiología, a physician from Cuba presented some roentgenograms in which the suprahepatic system was outlined partially by an opaque substance.

* Read before the Mexican Gastroenterological Association (Asociación Mexicana de Gastro-enterología).

FIG. 1. Suprahepatic circulation as seen after the injection of radiopaque material in an autopsy specimen of a normal liver.

In 1947, during the performance of an angiocardiography, the catheter accidentally slipped into the right suprahepatic vein and the injected opaque substance outlined the right portion of the suprahepatic system. On another occasion the inferior vena cava and part of the suprahepatic veins were made visible on the roentgenogram after the opaque substance was injected through a catheter introduced in the right auricle. These accidental findings suggested the possibility of: first, the roentgenological study of the suprahepatic system, and second, comparative chemical study (glucose, proteins) between peripheric venous blood and venous blood after its passage through the liver, an investigation that might bring light in the obscure field of hepatic metabolism.

We started these investigations in 1948, before the publication in the *Journal of Clinical Investigation* (March, September, 1949) of the papers by Billings and De Pree[1] and Bondy *et al.*[3]

We would like to point out briefly some anatomical concepts that are of great importance regarding this work, namely, the liver is a very vascular organ, the weight of the vessels and blood representing in normal conditions about 35 per cent of the total weight of the gland.[2,9] The blood reaches the liver by two different routes: the portal vein, and the hepatic artery which in spite of its small caliber contributes about 40 to 50 per cent of the oxygen requirements of the liver. These vessels branch off rapidly and when they reach the portal spaces, the portal vein ramifications penetrate the lobule and, uniting in the center of it, give rise to the central vein: the origin of the suprahepatic circulation.

The hepatic artery blood mixes with the portal vein blood in the sinusoids by means of arteriovenous communications within or outside of the lobule.

The suprahepatic system is formed by two different groups of veins: the first group is constituted by two veins, the right and the left, draining blood from the principal and accessory lobules; and the second group drains the territory near the vena cava.

Investigations in the liver of the frog have demonstrated that there are some areas where the hepatic circulation is very active, alternating with the areas of circulatory stasis. In normal conditions, about

FIG. 2. Catheter at the inferior vena cava, below the diaphragm. This vein, the right suprahepatic and some of their branches are clearly outlined.

75 per cent of the hepatic blood is in circulatory stasis.

Histological investigations have demonstrated that the suprahepatic veins have a peculiar arrangement of smooth muscle fibers forming sphincteric mechanisms that, as the one clearly seen in the ostium of the vena cava, regulate the outflow of blood from the liver.

Our personal work was done in three different phases: (1) study of the suprahepatic system in the cadaver; (2) catheterization and opacification of the suprahepatic system in the dog. Due to technical difficulties that have not been overcome completely, the films obtained are of a very poor quality and will not be presented; (3) study of the suprahepatic system in human subjects in whom we could not find signs or symptoms of liver disease.

Visualization and Anatomical Study of the Suprahepatic System in the Cadaver. The liver was removed from the abdominal cavity along with a segment of the inferior vena cava. The distal portion of the vein was ligated and through its proximal end one or two catheters were introduced into the right or left suprahepatic vein.

Fig. 3. Catheter right at the inlet of the right suprahepatic vein. It was possible to distinctly outline its three main branches.

Fig. 4. Catheter at the inferior branch of the right suprahepatic vein. The main trunks and all of their branches are shown.

A barium mixture was used as a contrast medium and the roentgenograms were taken in the anteroposterior projection.

The arrangement of the suprahepatic veins followed a constant pattern. We found in the roentgenograms obtained from the cadaver that there are two suprahepatic veins, the right and the left. The right one is formed by three main branches, the superior, middle and inferior. The left suprahepatic vein is formed by only two, superior and inferior.

We would like to call attention to the special way in which the collaterals open into the main branch. They do not form arborizations as is usually the case with other vessels, but small branches open directly into the main trunk making the vein look like a "branch with thorns."

The arrangement of the suprahepatic

FIG. 5. Catheter at the right suprahepatic vein. All the right suprahepatic system is clearly seen.

vessels in the diseased liver presents marked difference from that described for the normal gland. These studies will be the object of a future communication.

Visualization of the Suprahepatic System in the Living Subject and Comparative Anatomical Study with That of the Cadaver. The technique used for the catheterization of the suprahepatic veins in the living patient has been previously described by one of us (A. C.)[4,6] and, briefly explained, consists in the introduction of a Nélaton catheter in either left or right external jugular vein. The catheter is then advanced through the subclavian vein, superior vena cava, right auricle, inferior vena cava and, finally, into the suprahepatic vein. The catheterization of the suprahepatic veins sometimes is very difficult and the right vein is the one more frequently entered, possibly because of its greater diameter. When the catheterization of the suprahepatic veins proves impossible, the tip of the catheter is left in the inferior vena cava, immediately below the diaphragmatic dome, and the contrast substance injected right there. We use from 15 to 30 cc. of a 70–80 per cent nosylan solution injected as rapidly as possible.

We have used this technique satisfactorily in 30 cases in which the suprahepatic system has been made clearly visible.

During the study of these 30 cases we have encountered the following eventualities:

(1) The tip of the catheter lies in the inferior vena cava where the injection of the opaque substance is made, opacifying the inferior vena cava itself, the right and left suprahepatic veins and their main branches, giving a roentgen appearance exactly like that described for the liver of the cadaver.

(2) The catheter slips into either right or left suprahepatic vein and the opaque substance outlines that particular suprahepatic vein and all of its branches, showing the exact boundaries of the opacified vascular territory.

(3) When the catheter enters a secondary branch a homogeneous shadow corre-

FIG. 6. Hepatography of the inferior portion of the right lobe.

sponding to the opacified vascular territory is obtained. Roentgenoscopic control demonstrates that the shadow disappears completely within a few seconds. We consider that these apparently homogeneous shadows are the result of the superposition of minute shadows produced by the opaque substance in the capillaries, and not, as we first thought, by the spillage of contrast substance through a torn capillary. Not infrequently the injection of opaque substance in cases of this group resulted in pain that the patient felt in the right or left upper quadrant of the abdomen, depending on whether the right or left lobule had been catheterized. This pain persisted for a short time and subsequent clinical investigation failed to disclose any permanent damage of the liver.

(4) In some cases we observed that a new vessel, starting from the homogeneous shadow just described, was filled with opaque substance. This vessel branched off in exactly the opposite direction from that of the suprahepatic vessels. The collaterals of the former joined the main branch from downward up while the collaterals of the suprahepatic veins joined them from upward down. We believe that a branch of the portal system or from the hepatic artery was filled with the injected contrast substance through the suprahepatic capillaries and against the direction of the blood flow.

Fig. 7. Catheter at one of the branches of the left suprahepatic veins. Even opacity is seen corresponding to blood pools; probable initiation of the filling of the portal system.

Fig. 8. Catheter at one of the branches of the left suprahepatic vein, showing even opacity and, starting from there, progressively larger vessels with further ramifications are seen. We believe we were able, in this way, to outline the portal system.

This explanation is anatomically and functionally admissible since there is a proved continuity between the three circulations. Nevertheless we are inclined to believe that the opacified vessel is a portal branch, because: (*a*) the communications between the suprahepatic and portal veins are more numerous than those with the hepatic artery; (*b*) the blood pressure in the hepatic artery is greater than in the portal vein and it would be easier to counteract

the blood flow in the portal vein, and (*c*) circulation in the portal system is very slow.

If we are right in our assumption that the described vessel is an intrahepatic branch of the portal vein, we think that the opacification of a portal branch through the suprahepatic veins and against the blood flow in an individual without congenital anomalies has been achieved for the first time. The reactions experienced by the patients after the injection of the contrast substance were mild and consisted of a sensation of generalized warmth and a mild and transient pain in the right or left upper quadrant of the abdomen.

We believe that the contrast substance does not have any toxic effects on the hepatic cell, for (1) there has not been any clinical evidence of hepatic damage in any of the 30 cases studied. We propose to do hepatic function tests before and after hepatography in our next cases; (2) the macroscopic and microscopic study of the liver of a tuberculous patient, who died from a massive hemoptysis twenty-four hours after hepatography, demonstrated that the histologic structure of the gland was absolutely normal. This case might be regarded as experimental evidence of the innocuity of the contrast substance used; (3) in a case of cirrhosis of the Cruveilhier-Baumgarten type in which the portal circulation was made visible by the injection of nosylan into one of the collateral circulation veins,[5] no ill effects have been demonstrated three years after the study; in fact, the patient is very much relieved of his symptomatology, possibly because of the ligature of the umbilical vein after the introduction of the catheter.

In the present paper, we present the results obtained with the opacification of the suprahepatic circulation in normal individuals. The method has proved practical for the study of the anatomical distribution and characteristics of those vessels and, although only partially, for the study of the portal system. We are confident that the application of this method will be of assistance in the study of the pathological cases resulting in alteration of the hepatic vessels.

Hepatography in cases of liver cirrhosis has demonstrated profound alterations of the suprahepatic circulation. This study will be the object of a future communication.

SUMMARY AND CONCLUSIONS

A new method for the roentgenological study of the liver is described by means of the injection of a radiopaque substance into the suprahepatic veins. The following conclusions are reached:

1. The visualization of the suprahepatic system and of the parenchyma of the catheterized lobule is made possible by the above described procedure.
2. The opaque substance enters the portal vessels against the portal blood flow.
3. Subsequent studies will be necessary to judge the value of the procedure as a means of exploring the hepatic gland.
4. The catheterization of the suprahepatic veins and the injection of the opaque substance have proved harmless to the patient and to the liver in the thirty cases studied.

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THE EFFECT OF EXCLUSION OF THE BILE UPON GASTROINTESTINAL MOTILITY*†‡

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THE high incidence of disease involving the biliary tract has made this sytem the object of intensive study at the University of Pennsylvania. It has been estimated that 15 per cent of the adult population past forty years of age harbor gallstones,[56] and that operations upon the biliary tract are the second most frequent in number of laparotomies.[68] Upon empirical grounds, disease of the biliary system has long been considered an important cause of reflex disturbance of the gastrointestinal tract.[4,24,36] The clinical picture of the jaundiced patient with constipation was known to the ancient Egyptians who, as recorded in a papyrus of the year 1300 B.C., advised bile enemas.[29]

A new method for the study of the physiology of the gastrointestinal tract was reported in 1898 by Cannon.[20,23] This new method, which utilized the roentgen rays and a contrast meal, had the advantage that the motor phenomena of the gastrointestinal tract could be studied in the unoperated animal.[5] Since the time of Cannon's important contribution, the roentgen study of the gastrointestinal tract has been greatly extended. At the Hospital of the University of Pennsylvania much interest has centered upon the small intestinal pattern and motility as affected by various foods and drugs.[61,64] The response of the gastrointestinal tract to certain physical states (hyperthyroidism and adult tetany) has already been described.[60,64] The work of Barden, Ravdin and Frazier[6] on the effect of hypoproteinemia on the gastrointestinal motility,[55] and the work of Heublein, Thompson and Scully on the results of vitamin B_1 deprivation[47] were further extensions of the investigations into the physiology of the gastrointestinal tract. It was, however, felt that much remained to be learned about the effect of disease of the biliary system upon the gastrointestinal tract.

The present study is, therefore, the natural outcome of these two converging lines of investigation. It was agreed that the best approach to the problem was to observe the effect upon the gastrointestinal tract when the entire biliary secretion was prevented from entering the intestine although the complete absence of bile is not frequently met in clinical practice.

PART I. EXPERIMENTAL STUDIES

Method. A group of healthy dogs weighing 7.3 to 10.3 kg. was chosen, and subjected to a preliminary roentgen study of the gastrointestinal tract. The dogs at first were apprehensive and startled at the sounds emitted by the roentgen-ray apparatus, but soon learned to accept the noises as calmly as the leather straps which lightly bound their four legs to the corners of the roentgenoscopic table. As a further precaution against a possible psychic source of error, the straps were released when gastric emptying was slow in beginning. Some of the animals were so used to the position that they continued to lie on their backs after release of the straps.

The meal consisted of 2 parts of distilled water to 1 part barium. The volume was 225 cc. An ordinary rubber stomach tube was introduced and the barium meal injected. Roentgenoscopy was performed at quarter and half hourly intervals and roentgenographic records were made at intervals after roentgenoscopy to show, (1) the time at which the stomach began to empty, (2) the time for the stomach to empty itself, (3) the time required for the

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† This work was completed in 1939, but through error was not submitted for publication.

‡ Thesis submitted to the Faculty of the Graduate School of Medicine of the University of Pennsylvania, in partial fulfillment of the requirements for the degree of Doctor of Medical Science (D.Sc.[Med.]) for graduate work in Radiology.

TABLE I

PREOPERATIVE GASTROINTESTINAL SERIES

Dog No.	Stomach Begins to Empty	Stomach Empty*	Reaches Colon	Small Intestine Empty	Tone, Pattern, Remarks
95	35 min.	1 hr., 25 min.	3½ hr.	5 hr.	Stomach small, small intestine continuous stream, smooth ribbon, occasional longitudinal fold, some gas. Colon normal
175	15 min.	1¼ hr.	2¾ hr.	5½ hr.	Smooth ribbon with an occasional "string of wooden beads," some gas. Colon normal
177	40 min.	50 min.	50 min.	1⅓ hr.	Smooth ribbon, longitudinal folds, some gas. Colon normal
178	30 min.	2½ hr.	2½ hr.	5¼ hr.	Smooth ribbon in proximal portion. Many loops show coarsely stippled pattern. Colon normal
181	30 min.	30 min.	1½ hr.	4½ hr.	Smooth ribbon, longitudinal folds, gas. Colon normal
182	10 min.	1½ hr.	2¾ hr.	5¼ hr.+	Smooth ribbon, granular mottling, few folds, much peristalsis. Colon normal
186	15 min.	1 hr.	2 hr.	4¾ hr.	Smooth ribbon with occasional longitudinal fold, and gas. Colon normal
188	30 min.	2 hr.	2½ hr.	6½ hr.+	Stomach large. Small intestine, smooth ribbon with slight stippling and "string of wooden beads," gas. Colon normal
189	5 min.	1 hr.	2 hr.	4¾ hr.	Continuous ribbon, gas, no folds. Colon normal
190	5 min.	1¼ hr.	2 hr.	5¼ hr.	Smooth ribbon, some mottling, gas, an occasional longitudinal fold. Colon normal
192	5 min.	1 hr.	1½ hr.	5¾ hr.	Smooth ribbon with some very narrow loops, and rapid motility. Colon normal
193	10 min.	1¼ hr.	1¾ hr.	5½ hr.	Smooth ribbon and occasional mottled appearance of barium, gas. Colon normal
554	20 min. 14 min.	1 hr. 1¼ hr.	1¼ hr. 1¼ hr.	5¼ hr. 4¼ hr.	Smooth ribbon. Colon normal

* Expl. of Stomach—Since traces of barium were frequently found to remain in the stomach for hours, the time at which 90 per cent of the barium had left the stomach was used as a standard. This time was calculated from the moment when the first bolus left the stomach until 90 per cent of the barium was evacuated.

first bolus to reach the cecum, and (4) the length of time needed for the small intestine to empty itself of barium.

The results of the preliminary gastrointestinal examinations are summarized in Table 1. It can be seen that the preoperative emptying time of the stomach in every case was 2½ hours or less. The small intestinal passage time was measured by the interval required for the first bolus leaving the stomach to reach the cecum. This criterion was adopted to circumvent the occasional delay in the initiation of gastric emptying such as might be produced by emotional or extraneous causes. The range of values for small intestinal transit is also given in Table 1, and in 12 of 14 examinations was 2½ hours or less.

Observations were also made upon the pattern, caliber of small intestinal loops, presence of gas, and toxicity. The small intestine of the normal dog, as seen in the roentgen examination of the gastrointestinal tract, shows a continuous stream or "ribbon" of barium (Fig. 1, *1*). Frequently fine longitudinal folds and segmental contractions are visualized (Fig. 1, *3*). No transverse or "herringbone" appearances, such as typify the human small intestine, were seen. Occasionally, small intestinal gas was noted. These findings agree with the work of Barden, Ravdin and Frazier,[6] and Heublein, Thompson and Scully.[47]

Definitions. In the erect human subject, gastric tone is based upon the stomach's reception of the barium meal. In the dog, where intubation is practiced and the supine position used, it has been necessary to base the judg-

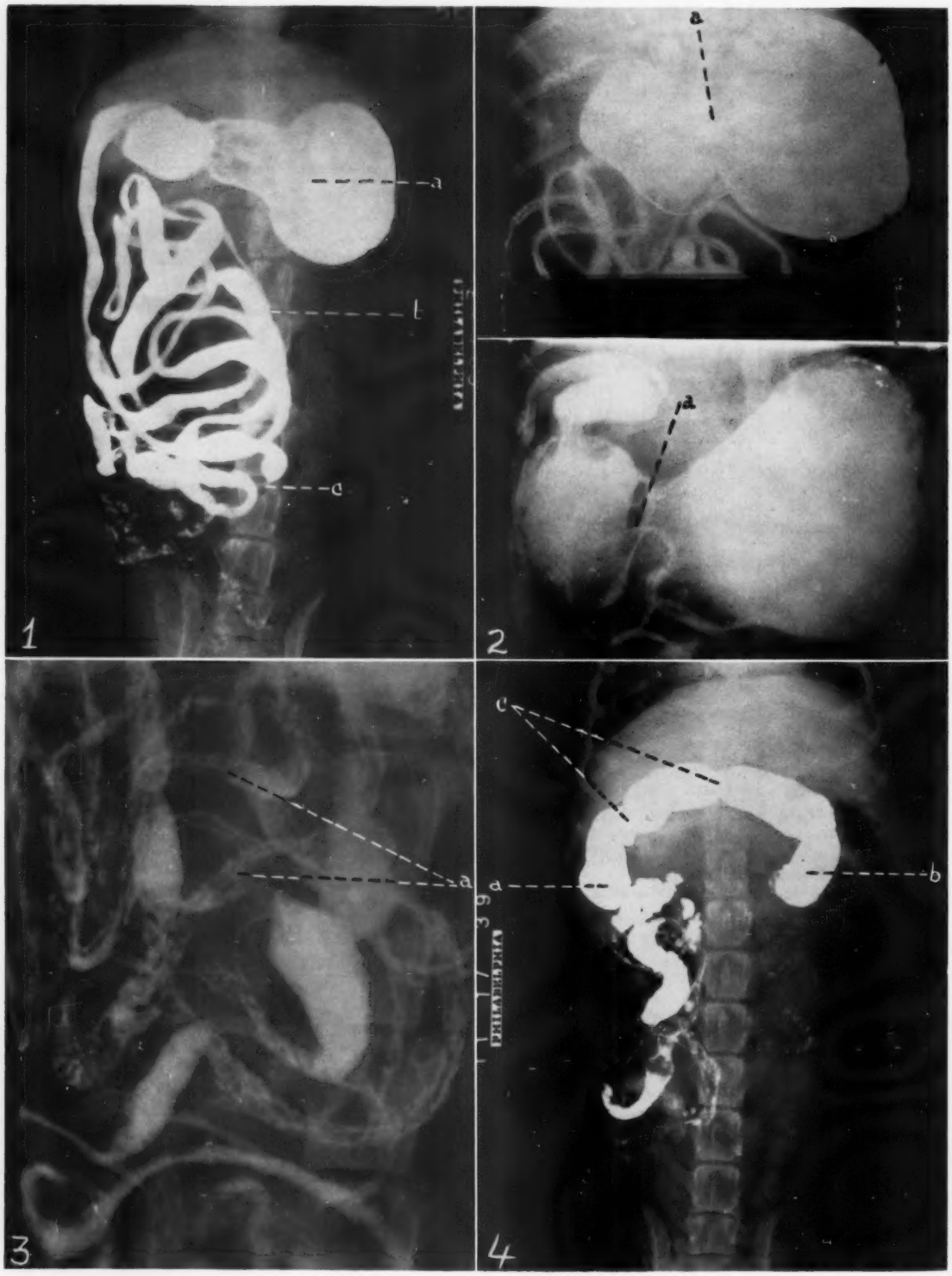


FIG. 1. (*1*) Dog No. 193. The gastrointestinal tract of the unoperated animal showing the stomach (*a*), the continuous "ribbon" of barium (*b*), with few segmental contractions (*c*).

(*2*) Dog No. 551. Showing *above* the stomach before exclusion of the bile, and *below* the stomach of the same animal after bile had been excluded from the gastrointestinal tract. A peristaltic wave is visible in each instance at (*a*). ⇛→

ment of tonus largely upon the size of the stomach. For this reason, a larger than normal stomach is taken to indicate a lowering of gastric tonus and, conversely, a stomach smaller than that of the control period is taken to indicate an increase in gastric tonus. A stomach of the control period is contrasted with a stomach of the period of exclusion of the bile (Fig. 1, *2*).

The small intestine of the "normal" dog shows a continuous ribbon of barium with occasional segmental contractions (Fig. 1, *1*). The mucosal pattern in the dog is not "herringbone" in type, but occasionally fine longitudinal folds are seen (Fig. 1, *3*). Where the caliber of the small intestine was wide, the intestine was considered to be hypotonic,[83] although it is realized that the only true determination of tonus is that made by the method of intubating with a balloon. Figure 2, *5* and *6*, illustrates these changes in caliber of the small intestine.

The width of the loops showed no correlation with the degree of segmentation or peristaltic activity, and there were numerous instances of increased segmentation and peristalsis in both wide (hypotonic) and narrow (hypertonic) small intestine.

To insure uniform conditions, the animals were given approximately the same amount of barium-distilled water mixture at each examination. Throughout the experiment they were maintained upon a mixed diet of kitchen scraps, low in fat. This latter provision was in view of the limited ability of animals (and humans) with common duct obstruction to absorb fats.[46,51] From time to time the animals received vitamins A and D orally, and subcutaneous injections of thiamin chloride (vitamin B_1). This was done in view of investigations which have shown that vitamins A and D are not readily absorbed from the bile-free intestine, and that animals with biliary fistulas have increased need of vitamins, presumably due to increased excretion of the vitamins through the fistula.[37,38,39] A deficiency of thiamin chloride (vitamin B_1) has been shown to produce a delay in gastrointestinal motility.[47]

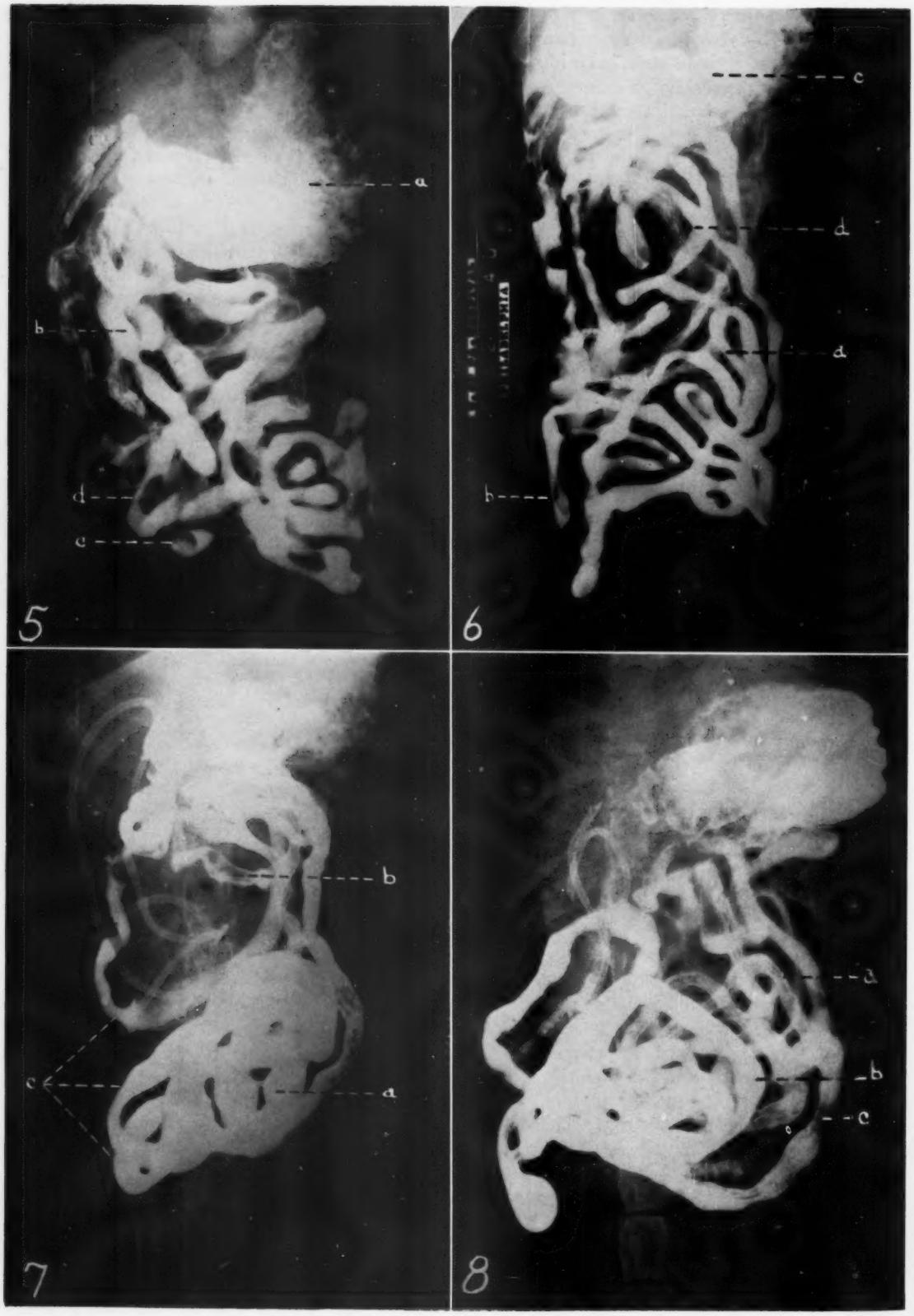
A large number of reports in the literature deals with the incidence of peptic ulcer in animals with complete exclusion of bile from the intestinal tract.[1,9,10,11,15,50,73,75,80,86] The incidence of such ulcerations in dogs with ligation of the common duct has been placed by some authors[15] at 60 per cent. This was an additional reason for the administration of vitamin A which has been shown to have a role in the protection of epithelial surfaces. Two further checks upon the formation of such ulcers were the repetition of the gastrointestinal examination *after* a period of bile exclusion, and the reintroduction of bile at intervals to prevent the formation of peptic ulcers. At necropsy, however, 3 of the dogs showed superficial ulcerations of the lower duodenum and jejunum. No gastric ulceration was found.

After the preliminary roentgen examination of the gastrointestinal tract had been made, the animals were subjected to operation.* A double intubation of the common duct was done, based upon a modification by McMaster and Elman[54] of the method devised by Rous and McMaster[70] for the sterile collection of bile. In this procedure the common duct is exposed and almost completely severed. One rubber catheter is inserted into the proximal (hepatic) end of the common duct, and made secure. The distal (duodenal) end of the common duct is intubated with a second catheter whose tip enters the duodenal lumen. The free ends of both catheters are brought out through the skin and joined to re-establish circulation of the bile until such time as the animal has recovered from the effects of the laparotomy. When it is desired to prevent the entrance of bile into the intestinal tract, the catheters are disconnected, and the bile draining from the catheter in the proximal portion of the common duct is collected in a sterile balloon. This balloon is encased for protection in a tin cylinder strapped to the animal's flank and the whole abdomen covered by a binder, so that the animal has no access to the catheters or biliary drainage (Fig. 3, *10*).

* All the surgical procedures were carried out by Dr. William Frazier.

(*3*) Enlarged photograph to show in detail the longitudinal folds (*a*) seen in the small intestine of the dog.

(*4*) Colon of the unoperated dog. The cecum is at (*a*) and the beginning of the descending colon at (*b*). Segmental contractions are visible at (*c*).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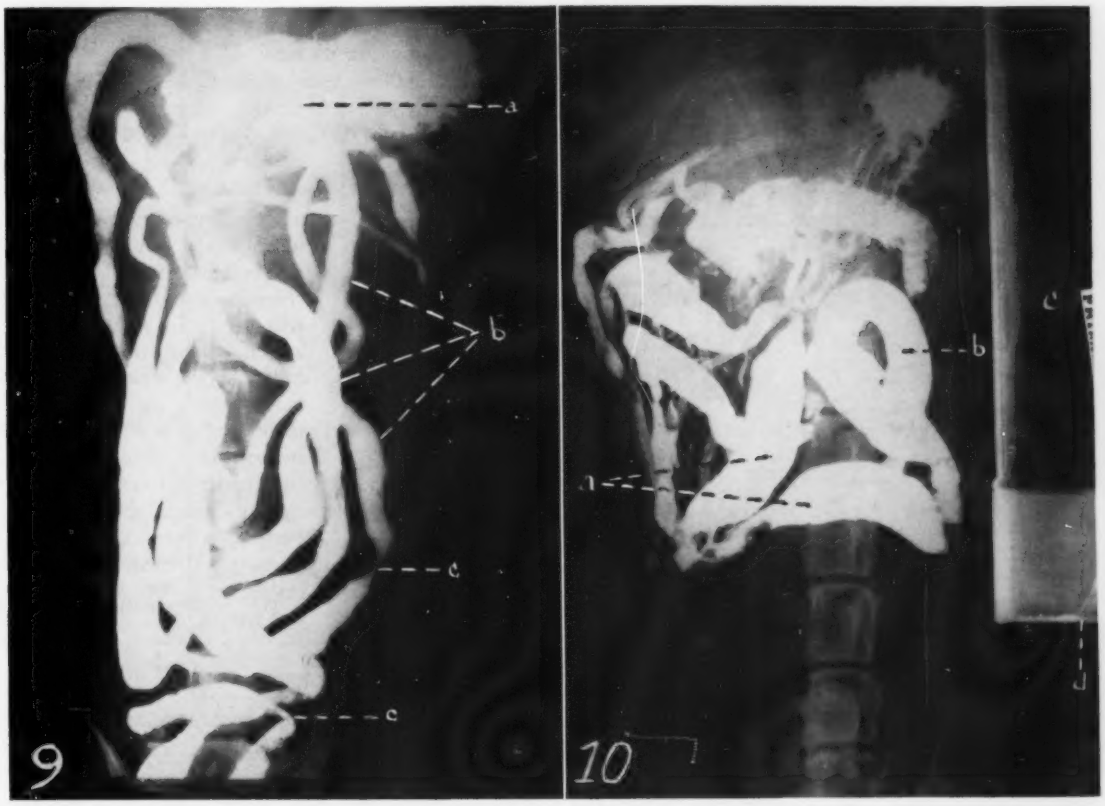


Fig. 3. (*9*) Examination after the administration of 0.5 gram of cholic acid. The stomach at (*a*) is of normal size. The small intestinal loops are widened and the meal is proceeding in a continuous stream (*b*). Segmental contractions are seen at (*c*).

(*10*) The bile-free intestine of the dog. Note the large sausage-shaped bolus at (*a*). The intestinal lumen is widened (*b*). The tin cylinder at (*c*) protects a balloon which collects the bile from catheter (*d*) in the hepatic end of the common duct.

After the bile had been excluded from the intestinal tract for at least one week, the roentgen examination of the gastrointestinal tract was repeated.* The findings are summarized in Table II, from which it is readily seen that the chief emphasis has been placed upon motor phenomena such as gastric emptying time, and period of small intestinal transit. These criteria were deliberately chosen because they had the virtue of being objective, and because early in the course of the experiments, it became evident that there was no uniform alteration of mucosal pattern, tonus, or contractile phenomena afforded by the bile-free gastrointestinal tract of the dog.

The chief responses noted after a period of exclusion of bile from the gastrointestinal tract

* At this time there were only 5 survivors of the original group of 12. Five of the dogs died before any surgical procedure was instituted. Two died shortly after operation from bile peritonitis.

Fig. 2. (*5*) Dog No. 95. Bile excluded from the intestine. The stomach (*a*) is large (hypotonic). The dilated small intestine (*b*) contains gas (*c*). The segmental contractions are visible at (*d*). The motility was delayed.

(*6*) Same dog. Examination after restoration of the bile to the intestine. The stream of barium was continuous and the caliber of the lumen narrow (*a*). There are few contractions visible (*b*). The stomach has returned to the preoperative size (*c*). A few longitudinal folds are visible at (*d*).

(*7*) Same dog. Examination made after including 1 gram of glycocholic acid in the barium meal. Note widened loops (*a*) and gas (*b*) in the small intestine. Continuous "ribbon" of barium (*c*). Very rapid motility.

(*8*) Examination made following the administration of 1 gram of taurocholic acid. The small intestinal loops tend to be wider than normal and the meal is proceeding in a continuous stream (*b*). Longitudinal folds are visible at (*a*), and gas and segmental contractions at (*c*).

TABLE II

GASTROINTESTINAL MOTILITY DURING EXCLUSION OF BILE

Dog No.	Stomach Begins to Empty	Stomach Empty	Time to Reach Colon	Small Intestine Empty	Tone, Pattern, Remarks
95	10 min.	3 hr., 5 min.	4 hr., 20 min.	6 hr., 20 min.+	Stomach hypotonic. Intestine wide, much gas, many contraction bands
175	20 min.	3 hr., 40 min.	1 hr., 10 min.	6¼ hr.	Stomach hypotonic. Delayed emptying. Intestine—narrow lumen, stream discontinuous. Many contractions. Caliber var.
178	45 min.	2¼ hr.	2¼ hr.	6¼ hr.	Intestine narrowed. Some loops show ribbon, others, the multiple bolus pattern
186	25 min.	1 hr., 35 min.	2 hr., 50 min.	5½ hr.	Loops widened, few contractions. Continuous stream
551	20 min.	4½ hr.	3½ hr.	6¾ hr.+	Continuous stream, much peristaltic activity, wide loops, and gas

are: (1) a tendency toward delay in the initiation of gastric emptying, (2) a delay in the gastric emptying time which, in one instance, reached three times the preoperative value, (3) a delay in small intestinal motility as measured by the time required for the first bolus to reach the cecum, and (4) increase in the time necessary for the small intestine to rid itself of barium. This agrees well with the work of Gutzeit and Kuhlbaum[43] and others.[3,44,45,72,81] Other workers,[2,17,18,25,29,35,57,74,79] using direct inspection by means of laparotomy, and preparations of surviving intestine, consider the presence of bile to inhibit motor function of the gastrointestinal tract. Other students of gastrointestinal physiology have found that the effect upon gastrointestinal motility is dependent upon the dosage of bile used, a large dose frequently reversing the effect of a smaller one.[12,42,59]

There was no uniform response to the absence of bile noted in the tonus or pattern of the gastrointestinal tract (Fig. 2, *5*). In 2 of 5 dogs, during the period of exclusion of the bile, the stomach was large (hypotonic). In 3, the small intestinal loops were wider (hypotonic). In 2 instances, the barium stream was no longer continuous. This is in accord with the observations of Bayer,[7,8] who considered a lowering of small intestinal tone as a diagnostic sign of hepatobiliary disease.

In order to rule out one complicating factor, namely, the operation by which the bile was excluded, the following studies were carried out. The two catheters were again connected and the flow of bile through the intestine re-established. After a minimum period of one week, the gastrointestinal examination was repeated. This third series of examinations was then compared with the second series of roentgen studies which was likewise performed after surgical intervention, the only difference between the two being the circulation of bile through the small intestine in the third series and its exclusion in the second series. These relationships are contrasted in Charts I to IV.

The four motor phenomena—(1) initiation of gastric emptying, (2) gastric emptying, (3) time to reach the cecum, (4) time to empty the small intestine of barium—seen while bile is excluded from the intestinal tract are contrasted with the examination made while bile was allowed to enter the intestine. As will be seen from Table III, the motility was delayed in nearly every instance during the period of bile exclusion. The changes in pattern and tonus during the period of restoration of bile to the intestine (Fig. 2, *6*) may be summarized briefly as a return toward the preoperative or control period.

Bile Acids. Sobotka, in his monograph on the bile,[77] lists among the substances to be found

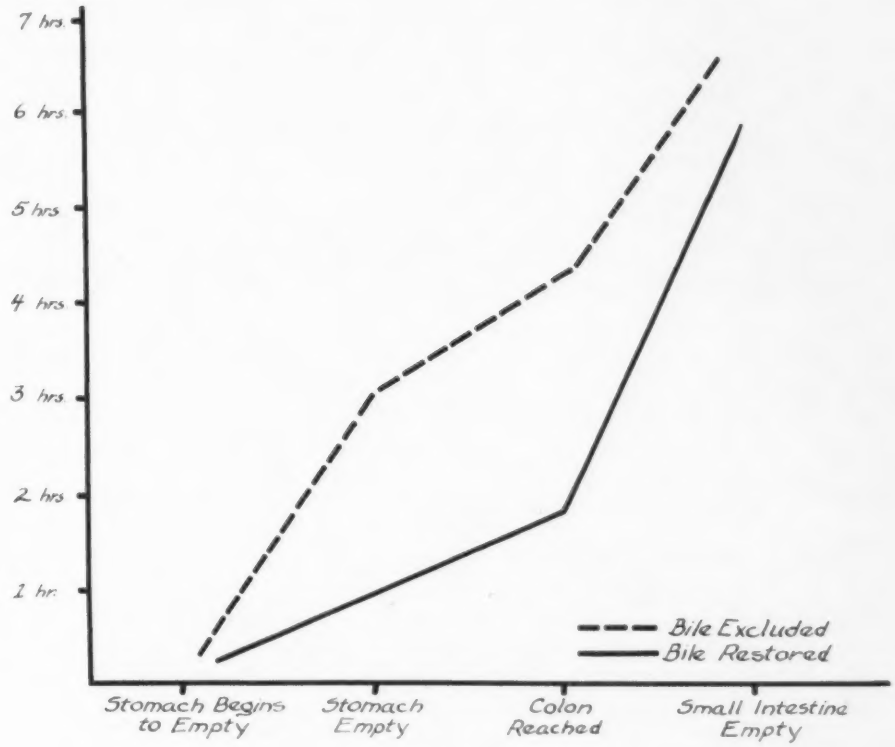


Chart I. Dog 95

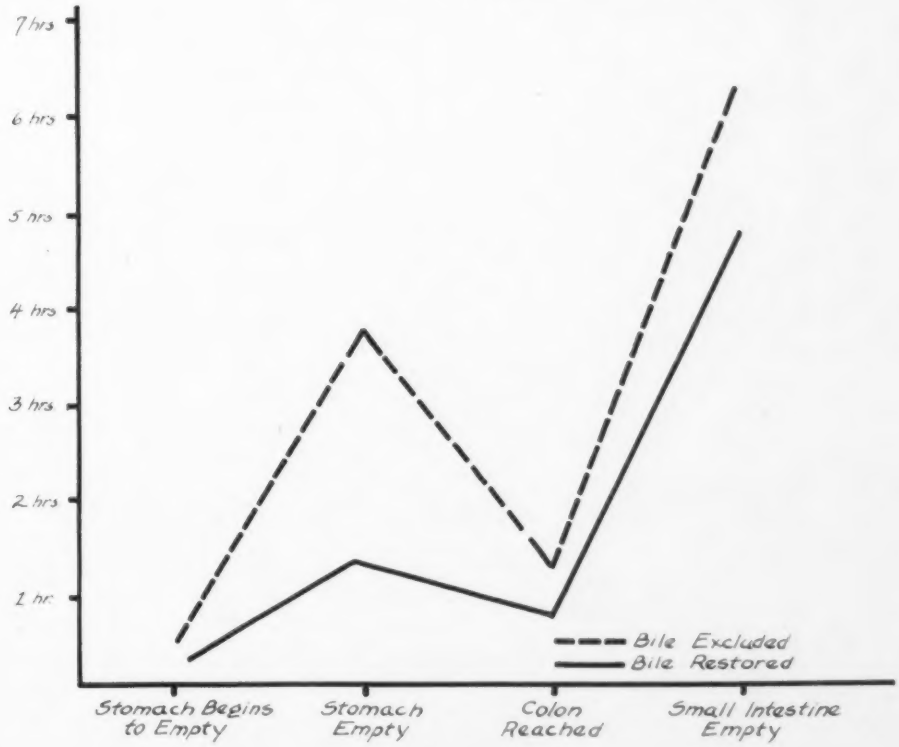


Chart II. Dog 175

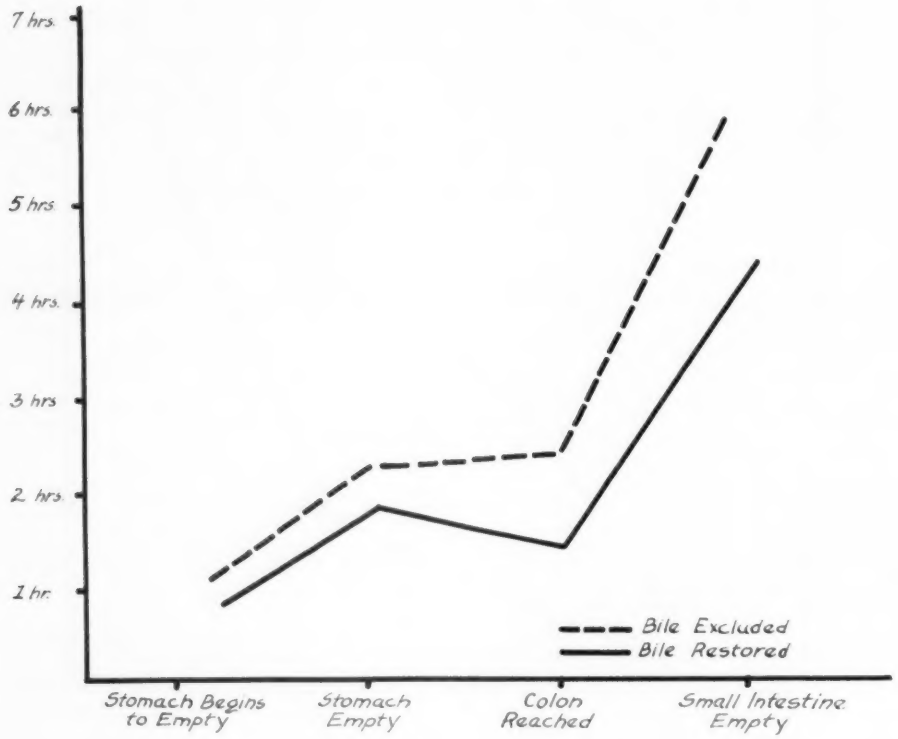


CHART III. Dog 178

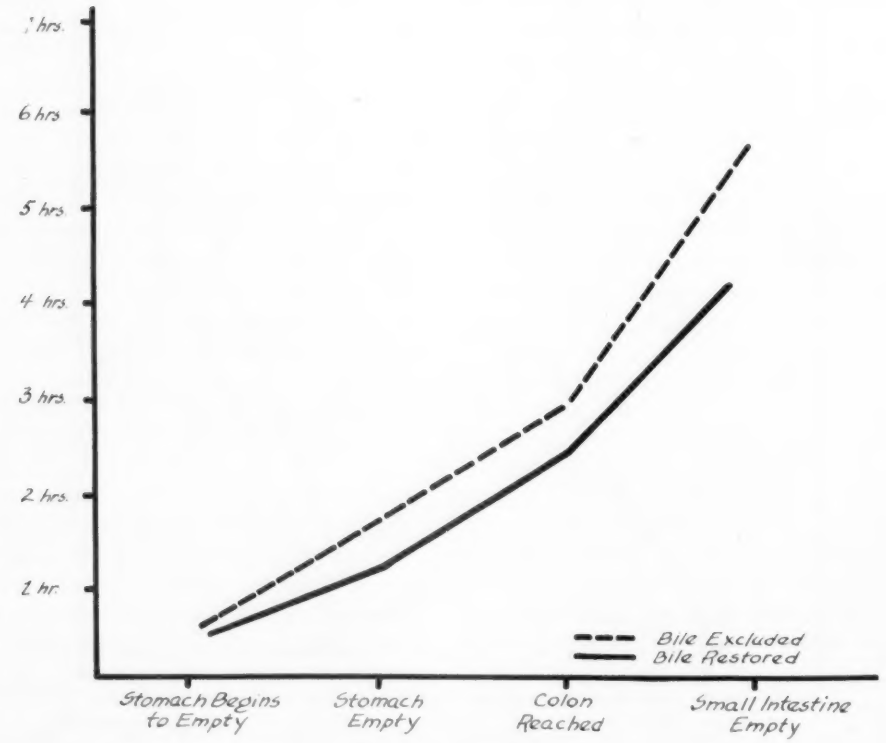


CHART IV. Dog 186

Table III

(1) INITIATION OF GASTRIC EMPTYING

Dog. No.	95	175	178	186
Bile excluded	10 min.	20 min.	45 min.	25 min.
Bile restored	10 min.	10 min.	30 min.	20 min.

(2) GASTRIC EMPTYING TIME*

Dog No.	95	175	178	186
Bile excluded	3 hr., 5 min.	3 hr., 40 min.	2¼ hr.	1 hr., 35 min.
Bile restored	1 hr.	1¼ hr.	1¾ hr.	1 hr., 10 min.

(3) TIME FOR FIRST BOLUS TO REACH COLON

Dog No.	95	175	178	186
Bile excluded	4 hr., 20 min.	1 hr., 10 min.	2¼ hr.	2 hr., 50 min.
Bile restored	1 hr., 50 min.	40 min.	1¼ hr.	2 hr., 25 min.

(4) TIME FOR SMALL INTESTINE TO EMPTY

Dog No.	95	175	178	186
Bile excluded	6 hr., 20 min.	6¼ hr.	6¼ hr.	5½ hr.
Bile restored	5 hr., 50 min.	4¾ hr.	4¼ hr.	4¼ hr.

* Since traces of barium frequently remained in the stomach for many hours, the time at which 90 per cent of the barium had left the stomach was used throughout the experiments.

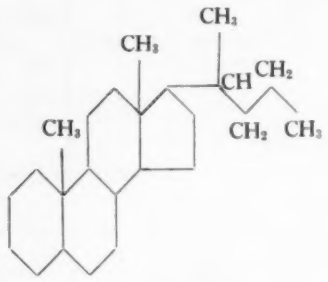
in the bile, (a) typical bile ingredients such as the alkali salts of the bile acids and bile pigments, (b) lipids, cholesterol being of special significance, (c) mucoprotein, (d) constituents common to all body fluids such as the *anions* Cl, HPO_4, H_2PO_4, CO_3, HCO_3 and the *cations*, Na, Ca, K, Mg, as well as CO_2, NH_3, urea, purine derivatives and enzymes.

The bile acids have been the object of considerable interest to workers in the field of hepatobiliary disease. Unlike the bile pigments which may be formed outside the liver cells, the bile acids seem to be formed only by the hepatic epithelium.[71] More recently evidence has been adduced to support the view that they are also destroyed by the liver.[16] Their chief function is to promote the absorption of fats from the intestine,[14,40,41,57,58,67] and deoxycholic acid is thought to be particularly concerned with keeping cholesterol in solution.[40,41,77] Cholic acid reduces the calcium content of the feces, presumably by promoting absorption of calcium from the intestine.[53,78] This is of interest in the light of the osteoporosis which is seen in some cases of biliary disease[48] and the zones of osteoporosis in the long bones of an infant with congenital atresia of the bile ducts reported by Caffey.[22] Bile acids are the most potent choleretics known.[32,65,84] Their ability to stimulate the secretion of bile has been noted roentgenoscopically, since the shadow of the gallbladder has been observed to increase in size one to two hours after the administration of sodium taurocholate.[85] The amount of bile salts secreted is influenced by the diet.[19,52,71] The disappearance of bile acids from the bile is a sensitive indicator of liver damage.[49,63,68]

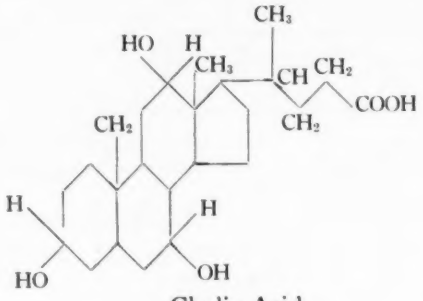
The type of bile acids secreted by an animal may be related to its diet and metabolic make-up.[77] Hog bile contains almost 100 per cent pure glycocholic acid, whereas the chief canine bile salt is taurocholic acid.[19,30,84] All fish belong to the taurocholic acid group. Cheno-(anthropo-) deoxycholic acid of human bile is the characteristic and predominant constituent of bird's bile.[77] Lithocholic acid is found in considerable amounts only in the bile of herbivores.[87]

The chief physiologic activities of the bile have been ascribed to its content of bile acids.[13,16,40,41,84,88] In man the chief bile acids are derivatives of a parent compound, cholanic acid.[71] They are cholic acid, deoxycholic acid, lithocholic acid,[86] and cheno- or anthropodeoxycholic acid. Only a minor percentage of the bile acids is found free in the bile, the major portion being conjugated with glycine or taurine.[26,71] For example, cholic acid combines with glycine to form glycocholic acid, and with taurine to form taurocholic acid.[33]

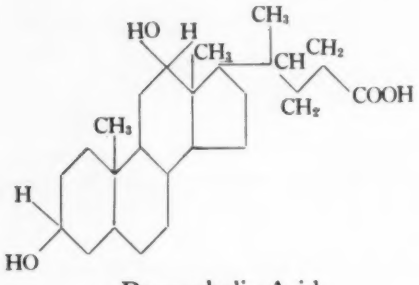
The bile acids have been shown to be of importance in the absorption of vitamins A and D[37,38,39] and vitamin K.[21,66,76] Structurally they are related to cholesterol,[40,86] ergosterol, to ovarian and testicular hormones, and to certain carcinogenic substances from coal tar.[41]



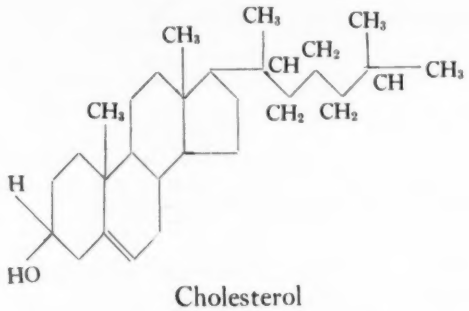
Cholane



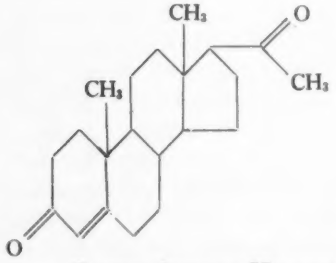
Cholic Acid



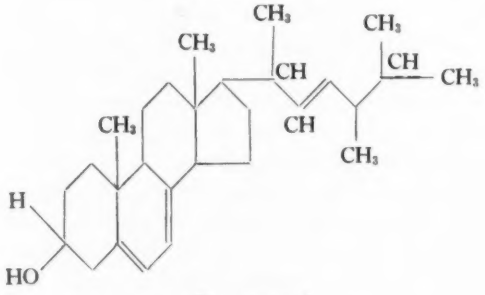
Deoxycholic Acid



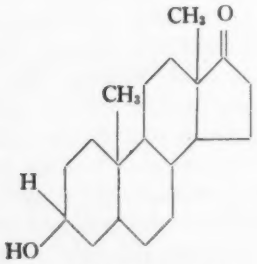
Cholesterol



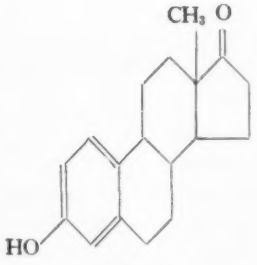
Corpus luteum Hormone



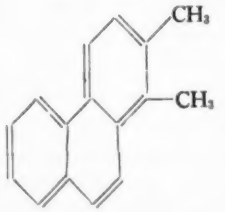
Ergosterol



Androsterone



Theelin



Dimethyl Phenanthrene

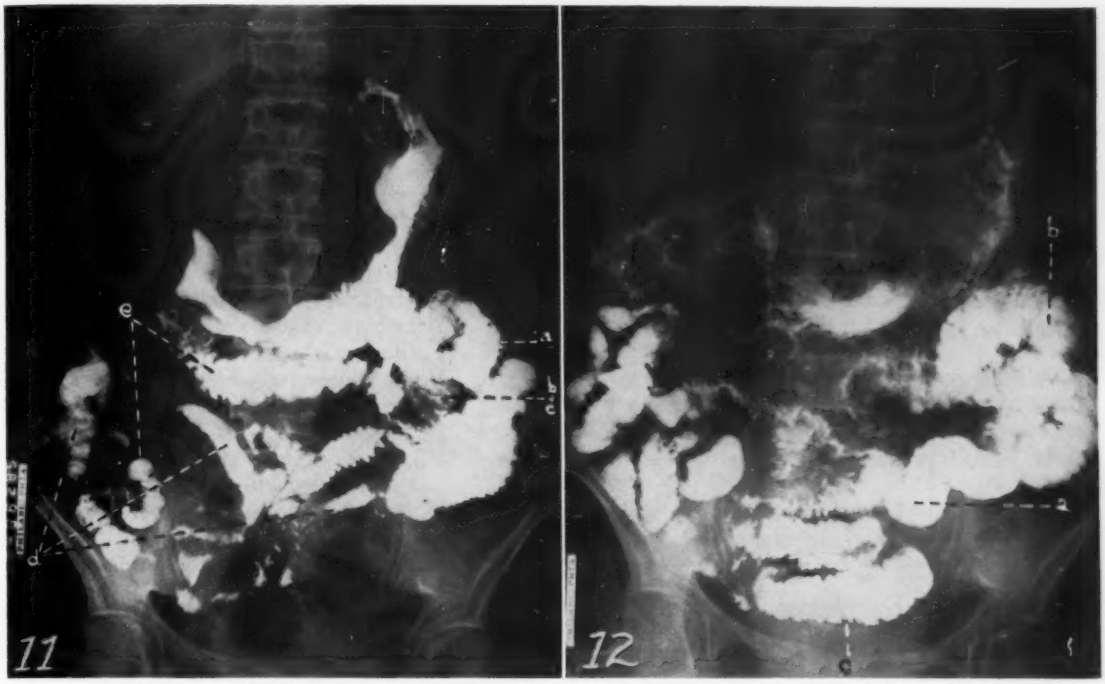


Fig. 4. (*11*) E.R. Preoperative examination. Film at 8¼ hours to demonstrate the wide (*a*), gas-filled (*b*) loops with transverse ridges (*c*) in place of the normal "herring-bone" pattern. The meal did not proceed in a continuous stream (*d*), and there was great variation in the caliber (*e*) of the loops.

(*12*) E.R. Same patient at 9½ hours. As yet, the meal has not reached the cecum. There is still further dilatation (*a*) of the loops, some of which contain gas (*b*). The mucosal pattern is coarse (*c*).

In an effort to discover which of the biliary constituents is responsible for the effect of bile on the propulsive motility of Thiry-Vella loops, Haney and co-workers[46] tested the effect of bile salts, bilirubin and biliverdin, Ca Cl_2, mucin and unsplit fats. They concluded that bile excites propulsive motility and "Of the several constituents of bile which were studied, only bile salts were found to have a similar effect. Thus the stimulating action of bile is readily accounted for on the basis of its bile salt content."[46]

For this reason it was considered desirable to observe the effect of various bile acids upon the gastrointestinal motility during the period of bile exclusion. Purified cholic, deoxycholic, glycocholic and dehydrocholic* acids were available, and a partially purified preparation of taurocholic acid.†

The bile acid to be tested (1 gram of taurocholic, glycocholic, and dehydrocholic acids, and 0.5 gram of cholic and deoxycholic acids) was mixed with the barium and water and introduced by a stomach tube in the ordinary manner, and the usual roentgen examination of the gastrointestinal tract carried out. The results with regard to motility can be summarized by saying that glycocholic acid appears to be the most potent accelerator of gastrointestinal activity. Next in order of potency comes sodium taurocholate, followed closely by deoxycholic acid. This agrees with the work of Schmidt and his co-workers,[71] who, in studying the volume output and bile salt content of fistula bile, found the conjugated bile acids more effective than the unconjugated bile acids in stimulating the increased secretion of solids in the bile, although dehydrocholic acid was also found by Riegel, Ravdin and Prushankin[69] to increase the output of cholic acid.

Doubilet[27,28] lists bile and bile acids in the order of their efficacy, as follows: canine bile acids, ox bile salts, glycocholic acid, cholic acid, deoxycholic acid and dehydrocholic acid.

The changes in pattern after administration of the bile salts varied from narrowed (hypertonic) small intestinal loops seen in 2 dogs after

* All courtesy of Riedel-de-Haen.
† Courtesy of Eastman-Kodak Company.

TABLE IV

DOG NO. 95. GASTROINTESTINAL MOTILITY FOLLOWING THE ADMINISTRATION OF BILE SALTS

Dog. No. 95	Stomach Begins to Empty	Stomach Empty	Time to Reach Colon	Small Intestine Empty	Tone, Pattern, Remarks
Glycocholic acid	Immed.	¾ hr.	3 hr.	4 hr.+	Stomach of normal size. Small intestine smooth ribbon. Wide lumen, many waves
Taurocholic acid	Immed.	1½ hr.	2 hr.	5 hr.	Stomach small. Small intestine smooth ribbon, caliber slightly increased, a few folds. Colon+
Deoxycholic acid	Immed.	1¼ hr.	3 hr.	5 hr.	Small intestine, wider lumen, few folds, many contractions visible, smooth ribbon
Cholic acid	Immed.	2 hr.	3¼ hr.	5½ hr.+	Small intestine, slightly enlarged, smooth ribbon, few folds and contractions
Dehydrocholic acid	Immed.	1¾ hr.	2½ hr.	6½ hr.+	Normal caliber, smooth ribbon, some longitudinal folds

dehydrocholic acid, to the wider (hypotonic) than normal loops seen in 4 of 5 dogs after glycocholic acid (Fig. 2, 7). The observations on dehydrocholic acid agree with the work of Freude[34] who found increased tonus after the use of this drug. The observations upon motor activity are tabulated in the individual protocols (Tables IV to VIII).

Discussion. The present series of experiments demonstrates that the exclusion of bile from the intestinal tract of the dog causes delay in gastrointestinal motility. Restoration of the bile stream to the intestinal lumen causes gastrointestinal motility to revert to normal. It would also appear likely that the motor activity of bile acids alone (*e.g.*, glychocholic acid) can cause gastrointestinal motility to increase and even exceed the preoperative motility. The mode of action of the bile acids is not definitely known. Among the hypotheses that have been advanced in the past are: (a) that bile acids act by virtue of their choleretic effect,[65,84] (b) that they act by promoting absorption of fats,[14,40,41,46,51] and (c) that they act directly on the intestinal

TABLE V

DOG NO. 175. GASTROINTESTINAL MOTILITY FOLLOWING THE ADMINISTRATION OF BILE SALTS

Dog. No. 175	Stomach Begins to Empty	Stomach Empty	Time to Reach Colon	Small Intestine Empty	Tone, Pattern, Remarks
Glycocholic acid	Immed.	1 hr.	1 hr.	3½ hr.	Intestine slightly wider, smooth ribbon, a little gas. No evacuation of colon in 3½ hr.
Taurocholic acid	Immed.	1 hr.	1 hr.	3½ hr.+	Stomach slightly larger. Intestine smooth ribbon. Not much gas. No evacuation of colon in 3½ hr.
Deoxycholic acid	5 min.	1¼ hr.	1¼ hr.	2¾ hr.+	Stomach large. Intestine, smooth ribbon, caliber slightly decreased
Cholic acid	5 min.	1¼ hr.	1¼ hr.	5 hr.+	Smooth ribbon, caliber slightly decreased
Dehydrocholic acid	1 hr.	3 hr.	2 hr.	5 hr.+	Stomach enlarged. Intestine, stream not continuous, lumen narrower, great variation in size

TABLE VI

DOG NO. 178. GASTROINTESTINAL MOTILITY FOLLOWING THE ADMINISTRATION OF BILE SALTS

Dog. No. 178	Stomach Begins to Empty	Stomach Empty	Time to Reach Colon	Small Intestine Empty	Tone, Pattern, Remarks
Glycocholic acid	15 min.	1 hr.	2¼ hr.	4½ hr.+	Stomach normal. Smooth ribbon. Colon normal
Taurocholic acid	Immed.	2¾ hr.	2½ hr.	6½ hr.+	Stomach small. Intestine, smooth ribbon caliber small early and normal later
Deoxycholic acid	10 min.	1 hr., 50 min.	1 hr., 50 min.	5 hr.	Intestine, smooth ribbon, narrow early and later normal width
Cholic acid	15 min.	1¼ hr.	1¾ hr.	5¼ hr.+	Stomach normal. Intestine, smooth ribbon, narrow early, and later of normal caliber. Colon slightly narrower
Dehydrocholic acid	45 min.	1¾ hr.	1¼ hr.	6 hr.+	Stomach small. Intestine, smooth ribbon, caliber narrow

wall,[8] or (d) upon the nervous mechanism.[8] While the present experiments do not point conclusively to the mechanism responsible for their action, they do tend to exclude hypothesis (a), for no bile was permitted to enter the intestinal tract during the period of the bile acid experiments. Hypothesis (b) is similarly unlikely to be the mechanism through which bile acids mediated their effect in the present series of investigations, for the diet was low in fat throughout the experiments. Hypothesis (c), which postulates an effect upon the intestinal wall, receives support from the present series of experiments, since 4 of 5 dogs showed widened (hypotonic) small intestinal loops following the administration of glycocholic acid. However, it must

TABLE VII

DOG NO. 186. GASTROINTESTINAL MOTILITY FOLLOWING THE ADMINISTRATION OF BILE SALTS

Dog. No. 186	Stomach Begins to Empty	Stomach Empty	Time to Reach Colon	Small Intestine Empty	Tone, Pattern, Remarks
Glycocholic acid	Immed.	45 min.	2 hr.	5½ hr.+	Stomach small. Intestine, smooth ribbon, a little gas, wider caliber. Colon large
Taurocholic acid	Immed.	2 hr.	2 hr.	5¾ hr.+	Intestine, continuous ribbon. Stomach and colon of normal size
Deoxycholic acid	Immed.	1½ hr.	3½ hr.	6¼ hr.+	Stomach, normal size. Intestine, smooth ribbon
Cholic acid	Immed.	1¾ hr.	2½ hr.	4½ hr.+	Stomach, normal. Intestine, smooth ribbon with gas, slightly larger. Colon normal
Dehydrocholic acid	20 min. 20 min.	1 hr., 25 min. 1 hr., 10 min.	4 hr., 10 min. 2 hr., 55 min.	5 hr., 40 min.+ 6 hr., 55 min.+	Stomach, large in first examination, smaller in second. Intestine, wide gas-filled loops, ribbon, some folds

TABLE VIII

DOG NO. 188. GASTROINTESTINAL MOTILITY FOLLOWING THE ADMINISTRATION OF BILE SALTS

Dog. No. 188	Stomach Begins to Empty	Stomach Empty	Time to Reach Colon	Small Intestine Empty	Tone, Pattern, Remarks
Glycocholic acid	Immed.	45 min.	1½ hr.	4½ hr.	Stomach normal. Intestine smooth ribbon, hypotonic
Taurocholic acid	10 min.	1 hr., 50 min.	1 hr., 50 min.	5 hr., 50 min.+	Stomach normal. Intestine smooth ribbon, normal caliber, stippling of barium
Deoxycholic acid	7 min.	1½ hr.	2¾ hr.	4½ hr.+	Stomach normal. Intestine, smooth ribbon with gas
Cholic acid	20 min.	1 hr., 40 min.	1 hr., 40 min.	4 hr., 55 min.+	Stomach normal. Intestine, smooth ribbon with "beading" of barium. Colon normal
Dehydrocholic acid	15 min. 15 min.	2½ hr. 2½ hr.	2½ hr. 2½ hr.	6 hr.+ 3¾ hr.	Stomach normal. Intestine, smooth ribbon, narrow early and wider later, some gas. Colon normal

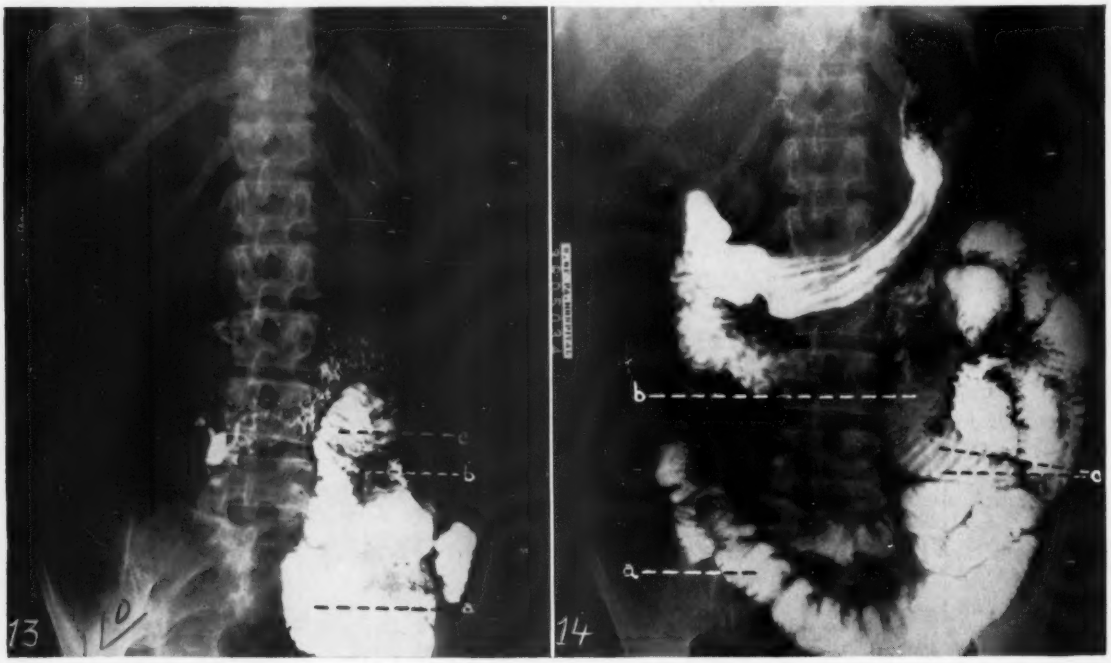


FIG. 5. (*13*) N.S. Preoperative examination. Roentgenogram taken at 2½ hours to show dilated loops of small intestine (*a*) containing gas (*b*). "Moulage" type of pattern (*c*). Delayed motility.

(*14*) N.S. Postoperative examination. Roentgenogram taken at 1½ hours showing an increase in the dilatation of the loops (*a*). The gas-filled loop (*b*) in the left upper quadrant with mucosal folds extending completely across (*c*) is a typical finding.

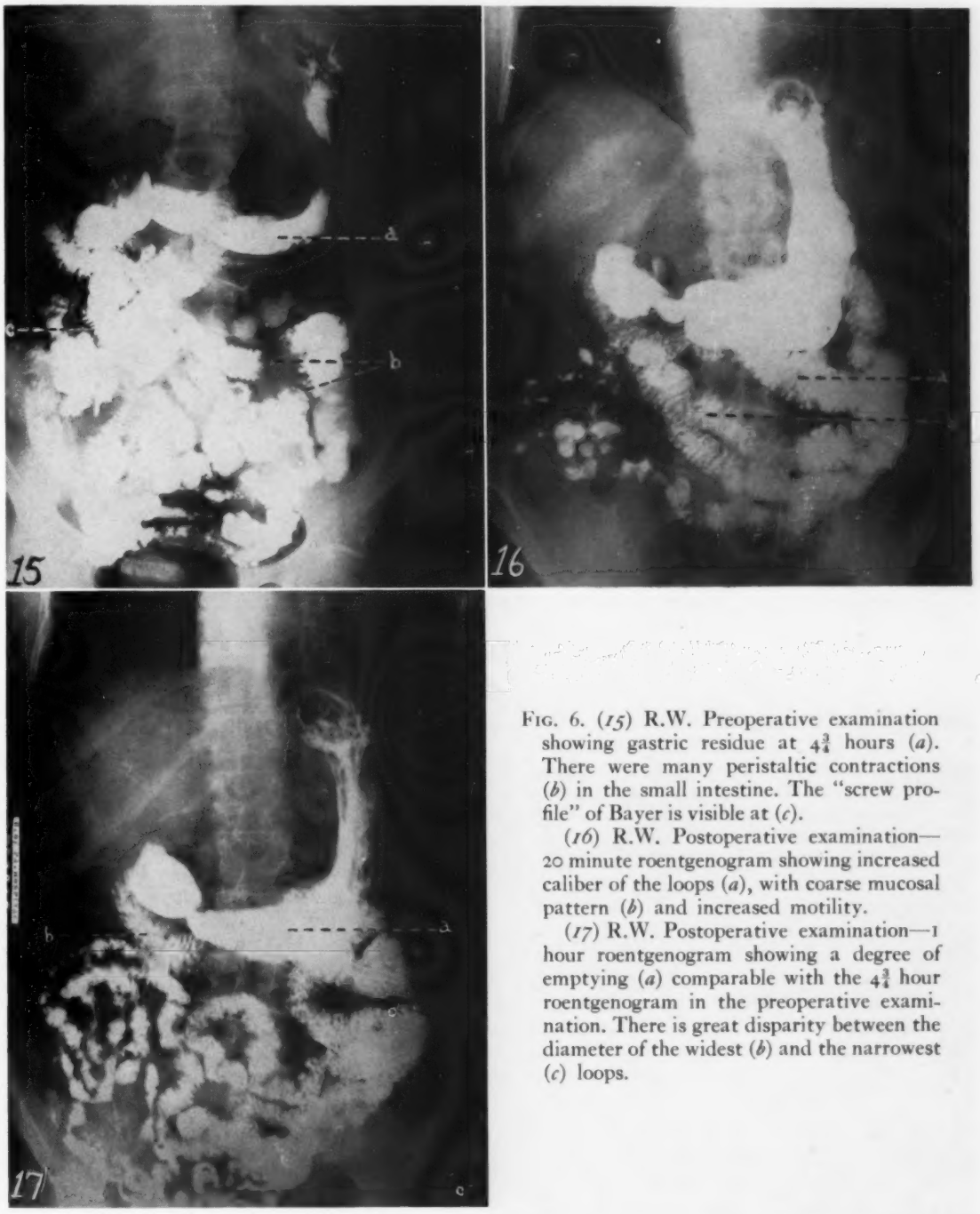


Fig. 6. (*15*) R.W. Preoperative examination showing gastric residue at $4\frac{3}{4}$ hours (*a*). There were many peristaltic contractions (*b*) in the small intestine. The "screw profile" of Bayer is visible at (*c*).

(*16*) R.W. Postoperative examination—20 minute roentgenogram showing increased caliber of the loops (*a*), with coarse mucosal pattern (*b*) and increased motility.

(*17*) R.W. Postoperative examination—1 hour roentgenogram showing a degree of emptying (*a*) comparable with the $4\frac{3}{4}$ hour roentgenogram in the preoperative examination. There is great disparity between the diameter of the widest (*b*) and the narrowest (*c*) loops.

be concluded that none of the hypotheses advanced satisfies all the known facts.

Summary. Total or even partial exclusion of bile from the gastrointestinal tract of the dog retards gastric emptying and small intestinal motility. Restoration of the bile stream causes the gastrointestinal motility to revert toward the normal. Bile acids can be substituted for whole bile in influencing the gastrointestinal motility. There is no typical pattern or appearance of the gastrointestinal tract of the dog during the period of exclusion of the bile.

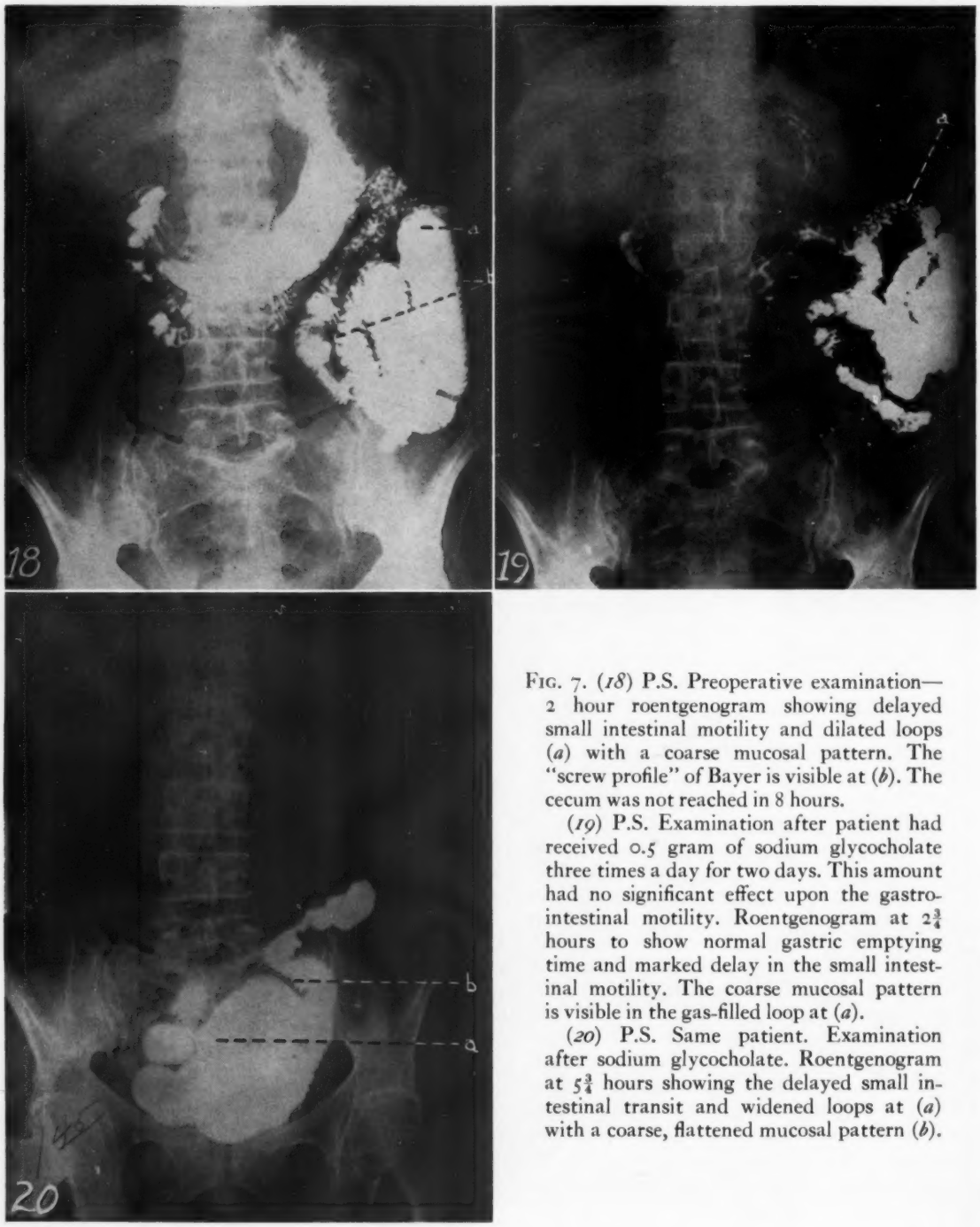


Fig. 7. (*18*) P.S. Preoperative examination—2 hour roentgenogram showing delayed small intestinal motility and dilated loops (*a*) with a coarse mucosal pattern. The "screw profile" of Bayer is visible at (*b*). The cecum was not reached in 8 hours.

(*19*) P.S. Examination after patient had received 0.5 gram of sodium glycocholate three times a day for two days. This amount had no significant effect upon the gastrointestinal motility. Roentgenogram at $2\frac{3}{4}$ hours to show normal gastric emptying time and marked delay in the small intestinal motility. The coarse mucosal pattern is visible in the gas-filled loop at (*a*).

(*20*) P.S. Same patient. Examination after sodium glycocholate. Roentgenogram at $5\frac{3}{4}$ hours showing the delayed small intestinal transit and widened loops at (*a*) with a coarse, flattened mucosal pattern (*b*).

PART II. CLINICAL STUDIES

There have been few reports of roentgen studies of the acholic gastrointestinal tract, probably for the reason that the diagnosis of common duct obstruction was evident and the further reason that the patients in most instances were too ill to submit to an examination that might extend over a period of eight hours or more.

Bayer[7,8] observed delayed gastric empty-

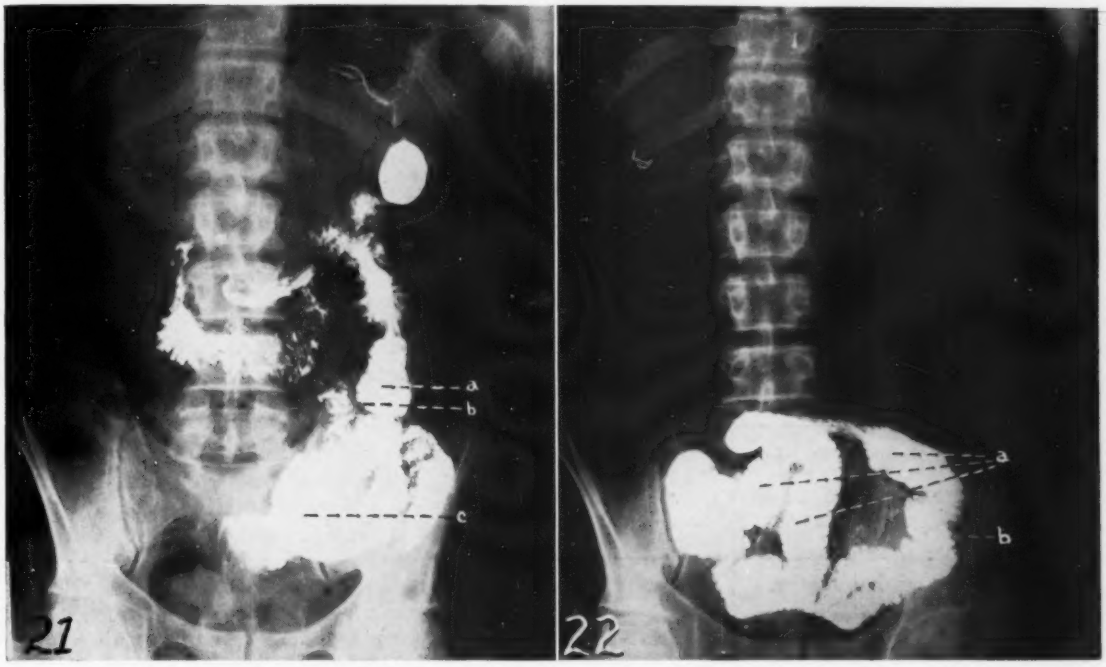


Fig. 8. (*21*) C.L. Preoperative examination—2¾ hour roentgenogram showing wide loops (*a*) with coarse mucosal pattern (*b*). There was stasis in the jejunum (*c*).

(*22*) C.L. Same patient at 8½ hours, showing the dilatation (*a*) of the small intestine, the coarse mucosal pattern (*b*), and the delayed motility. The meal has not yet reached the cecum.

ing (the exact composition of the meal not given), delay in the small intestinal passage time, and hypotonus in a patient with carcinoma of the gallbladder and acholic stools. The mucosal folds were flattened, which the author ascribed to an effect upon tonus. The same worker believed that the delayed passage time was not due to inhibition of peristalsis but to atony of the intestine which was toxic or even cholemic.

Gutzeit and Kuhlbaum,[43] who performed roentgen examinations upon the gastrointestinal tract of patients with complete and nearly complete acholia, demonstrated marked delay in the transit of the barium meal through the small intestine. The exact composition of the meal was not stated. In one patient, the time required for the meal to pass from the stomach to the cecum was 7½ hours, while another patient, after 9½ hours, still presented one-half the meal in the small intestine. Both patients exhibited considerable increase in the caliber of the small intestine. The authors ascribed the changes in motility and pattern to alterations in both peristalsis and tonus.

Freude,[34] making roentgenoscopic observations in a patient with pylorospasm after the injection of a 20 per cent solution of dehydrocholic acid, found relaxation of the spasm, increased speed of gastric emptying, and increased tonus.

Boulet, who had observed inhibition of spontaneous contractions of the surviving intestine of the rabbit, hog, sheep and dog,[17] when bile was placed in contact with either the mucosal or serosal surface, was able to obtain segments of both large and small intestine from two men shortly after they had been decapitated.[18] His experiments are sufficiently unusual to warrant more detailed attention. With the temperature of the solution raised to body heat, the response of the duodenum to 3 cc. of

TABLE IX

GASTROINTESTINAL MOTILITY IN SEVEN PATIENTS WITH COMMON DUCT OBSTRUCTION

Patient	Stomach Begins to Empty	Stomach Empty*	Reaches Colon	Small Intestine Empty	Tone, Pattern, Remarks
C.O'H. Case 1	Immed.	5 hr.	Not in 8 hr.	24 hr.	Stomach normal tonus, gas in small intestine. Continuous stream early, later column broken up. Coarsened mucosal pattern. Normal caliber. R.L.Q., many peristaltic contractions
E.R. Case 2	Few min.	9½ hr.	Not in 9½ hr.	Before 24 hr.	Stomach hypotonic and with hypoperistalsis. Small intestine, gas. Wide loops with coarse mucosal pattern. Great variation in caliber
C.L. Case 3	Few min.	2¾ hr.	Not in 9½ hr.	Not in 9½ hr.	Stomach showed active peristalsis. Small intestine loops widened with coarse mucosal pattern and many peristaltic contractions. Stasis in the jejunum
R.A. Case 4	Few min.	3½ hr.	7½ hr.	Not in 8 hr.	Stomach of normal tone. No residue. Normal peristalsis. Jejunum and ileum widened with coarse mucosal pattern and gas filled loops. Continuous stream. Many peristaltic contractions visible
P.S. Case 5	½ hr.	2½ hr.	Not in 8 hr.	Not in 8 hr.	Stomach—hypotonic and in diastole. Small intestine loops wide with coarse mucosal pattern. Continuous stream of barium
R.W. Case 6	Few min.	Not in 4¾ hr.	Not in 4¾ hr.		Gastric peristalsis hypoactive. Loops normal caliber with many peristaltic contractions
N.S. Case 7	Immed.	2½ hr.	Nearly in 5 hr.	About 7 hr.	Hyperactive gastric peristalsis with commensurate motility. Prox. small intestine dilated with gas filled loops and motility was slowed. Continuous stream. "Moulage" type of pattern. Colon—increased prominence of mucosal pattern.

* Since traces of barium were frequently found to remain in the stomach for hours, the time at which 90 per cent of the barium had left the stomach was used as a standard. This time was calculated from the moment when the first bolus left the stomach until 90 per cent of the barium was evacuated.

bile in 30 cc. of saline solution was recorded. An immediate increase in tonus was observed which reached a peak in three minutes and then slowly returned to normal at the end of eleven minutes. The bile used was that of the experimental subject.

The response of the ileum to bile was one of relaxation and suppression of contractions. There was no return to the initial tonus until lavage of the segment was practiced. The movements reappeared and were more vigorous than before. The application of bile once more caused the contractions to disappear.

From a recent group of 50 cases of common duct obstruction, including calculus, treated in the Surgical Department of the Hospital of the University of Pennsylvania, only 7 patients could be selected who had had a preoperative roentgen study of the gastrointestinal tract. A brief summary of each case is presented, with the salient points in the roentgen study and the data summarized in Table IX. It would perhaps

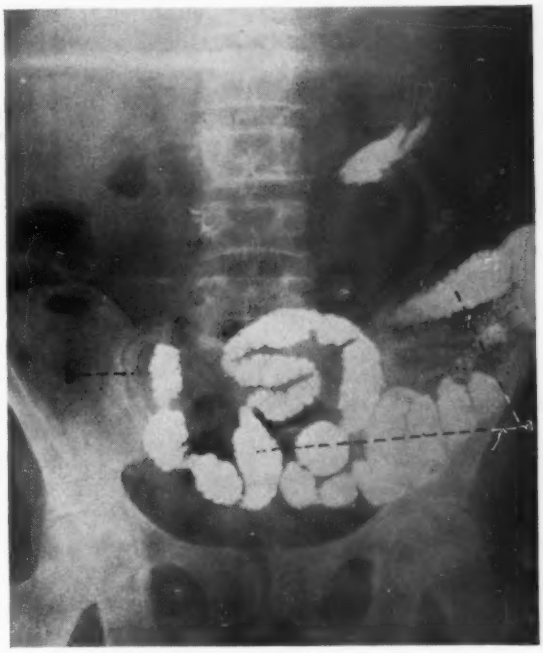


Fig. 9. R.A. Preoperative examination. Roentgenogram at 3¾ hours showing widened jejunal and ileal loops (*a*), with gas and coarse mucosal pattern (*b*). Motility was delayed.

be well to state the criteria of the "normal" gastrointestinal examination, before proceeding to the deviations from the standard which were observed in the patients to be reported.

The meal used in every instance consisted of 5 oz. of barium sulfate, thoroughly dispersed (by an electric mixer) in 5 oz. of distilled water. Gastric tonus was determined by the stomach's reception of the water-barium meal. If the first mouthful of barium proceeded smoothly down the lesser curvature, or magenstrasse, the stomach was considered orthotonic. If the barium plummeted down to the most dependent portion of the stomach, the stomach was considered hypotonic. In those cases where the barium was held up in the juxta-esophageal portion of the stomach hypertonicity was considered to be present.

The "normally" active stomach presents three to four peristaltic contractions per minute. The waves may be of the deeply segmenting type or may indent the curvatures to a lesser degree. The presence of gas in the lower small intestine of the adult is considered abnormal.

The normal small intestinal pattern presents the characteristic "herringbone" arrangement of mucosal folds in the jejunum. The ileum is characterized by the bolus pattern and the "stack of coins." Not infrequently, longitudinal folds are seen, particularly in the terminal ileum. The meal proceeds in a continuous stream and peristaltic rushes are considered abnormal.

The water-barium meal as described above was used throughout the examinations reported below.

Case 1. C.O'H., a clinic patient, aged sixty-seven, who had for several years had bouts of

Table X

Patient R.W.	Stomach Begins to Empty	Stomach Empty	Reaches Colon	Small Intestine Empty	Tone, Pattern, Remarks
Preoperative	Few min.	Not in 4¾ hr.	Not in 4¾ hr.		Gastric peristalsis hypoactive. Loops normal caliber with much evidence of peristaltic contractions
Postoperative	Few min.	3 hr.+	2½ hr.	Nearly in 6¾ hr.	More active gastric peristalsis. Loops broader than preoperative and mucosal pattern coarser. Great disparity between largest and smallest loops. Early stream of barium continuous, later discontinuous. Some gas-filled loops. Much peristaltic contraction

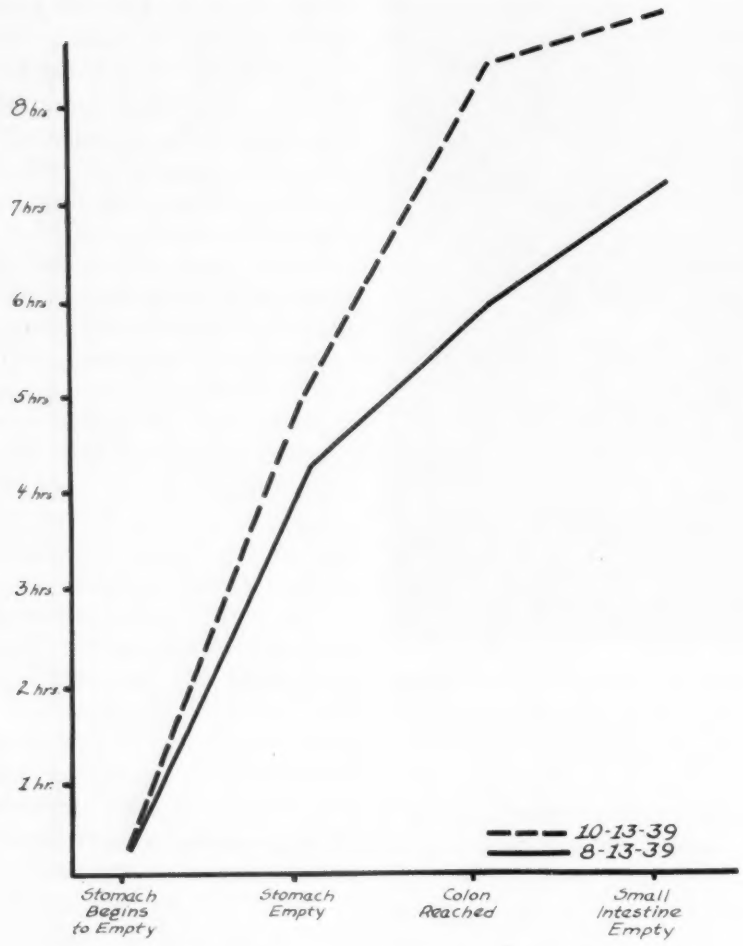


CHART V. C.O'H. Progressive slowing of gastrointestinal motility.

painless jaundice, particularly following alcoholic debauches. A gastrointestinal examination on August 13, 1939, showed marked hypomotility of the small intestine. The gastric emptying time was $4\frac{1}{4}$ hours, the cecum appearance time was six hours and the small intestine was not empty in seven hours. Puddling of barium was seen.

On August 30, 1939, the patient was admitted to the ward service of Dr. Ravdin, with a chief complaint of being "yellow." The stools were not acholic. Cholecystostomy was performed on August 31, 1939, and calculi removed. The biliary drainage never showed bile salts, and a cholangiogram on September 25, 1939, demonstrated blockage of the common duct.

A repetition of the gastrointestinal examination on October 13, 1939, showed an increased delay in the gastrointestinal motility so that the stomach now emptied in five hours and the column had not reached the cecum in eight hours. The small intestine was not empty in eight hours. The small intestinal loops were widened and the mucosal pattern was coarsened (Fig. 10). The two examinations are presented in Chart v.

The patient became asthenic and died on October 27, 1939. Autopsy showed carcinoma of the head of the pancreas with obstruction of the common duct secondary to fibrosis of the head of the pancreas.

CASE 2. E.R., aged sixty-eight. Admitted to Dr. Ravdin's service on October 6, 1939 with a chief complaint of indigestion and pain in back. Patient had sudden onset of jaundice in September, 1939, after an attack of colicky pain. This jaundice disappeared after one week but returned and became increasingly deep. Patient has occasionally observed clay-colored

stools. On admission, no bile could be obtained on biliary drainage. The stools were not clay-colored. The indirect Van den Bergh was 20; protein, 6.8 gm.

Gastrointestinal examination performed on October 13, 1939, showed marked delay in the gastric emptying, some barium remaining 9½ hours after ingestion. The small intestine showed marked hypomotility with dilated, gas-filled loops, and the cecum was not reached in 9½ hours (Fig. 4, *11* and *12*).

Autopsy revealed calculous cholecystitis with stone in the common duct and carcinoma of the head of the pancreas.

Case 3. C.L. Admitted to the service of Dr. Ravdin on October 21, 1939, with a chief complaint of aching pain in the right upper quadrant for two months. On admission, the patient was jaundiced and found to have diabetes mellitus. The presumptive diagnosis was carcinoma of the ampulla of Vater with obstructive jaundice. The Van den Bergh reaction showed an immediate direct reaction, and 34 units indirect. The cholesterol was the highest ever recorded in the hospital laboratory, 1,000 mg. per cent.

Gastrointestinal examination on December 14, 1939, showed normal gastric motility with a marked delay in small intestinal transit. The loops were widened and showed coarse mucosal folds and many segmental contractions. There was stasis of barium in the jejunum. The barium did not reach the cecum in 9½ hours. The changes are shown in Figure 8, *21* and *22*. Because the roentgenogram of the chest suggested the presence of metastatic malignancy, surgery was not done.

Case 4. R.A., aged fifty-nine. Admitted to the service of Dr. Eliason on October 12, 1939. The diagnosis of obstructive jaundice was evident and the Van den Bergh reaction showed direct positive, indirect 28–35.2 units.

Gastrointestinal examination on October 17, 1939, showed a somewhat delayed gastric emptying and a definitely delayed small intestinal motility. The meal reached the cecum in 7½ hours. The small intestine showed wide, gas-filled loops and many peristaltic contractions (Fig. 9).

At operation on October 20, 1939, a hard, distended gallbladder was visualized and there were nodules in the liver which, on frozen section, proved to be adenocarcinoma.

Fig. 10. C.O'H. Roentgenogram made at 1¾ hours showing coarse mucosal pattern at (*a*).

The patient died on October 31, 1939, and autopsy was refused.

Case 5. P.S., aged sixty-five. Admitted to the Graduate Hospital on May 11, 1937, where a cholecystectomy was performed and a stone removed from the common duct. At the time of admission to the Graduate Hospital, the patient was not jaundiced. A diagnosis of diabetes mellitus was made. In the months preceding her admission to the University Hospital, the diabetes mellitus had been uncontrolled, and had resulted in a weight loss of 40 pounds. On admission to the University Hospital, the patient was mildly jaundiced, and the degree of jaundice increased during her sojourn in the hospital. The Van den Bergh reaction rose from 2 units to 7 units. There was marked anemia (red blood cell count, 1.8 million; Hb, 33 per cent).

A gastrointestinal examination was performed on April 12, 1940 to bring to light a possible pancreatic lesion. There was a delay in the initiation of gastric emptying so that very little barium left the stomach during the first half hour. The stomach, however, emptied in normal time. There was marked delay in the transit through the small intestine and the

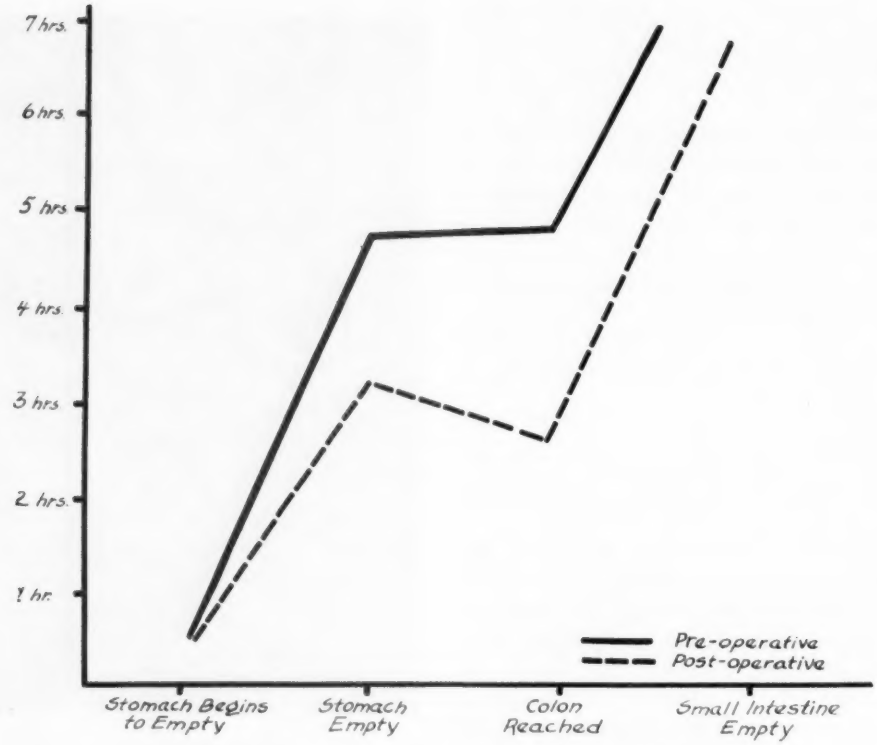


CHART VI. R. W. Gastrointestinal motility before and after operation.

cecum was not reached in eight hours. The loops of the small intestine were wide and showed a coarse mucosal pattern (Fig. 7, *18*).

The examination was repeated after the patient had received 0.5 gram of sodium glycocholate three times a day for two days. There was no change in the motility or pattern in this short time. The findings are presented in Figure 7, *19* and *20*.

CASE 6. R.W., aged sixty-three. Admitted to the service of Dr. Ravdin on March 28, 1938, with a five months' history of nocturnal attacks of right upper quadrant pain. The stools had become increasingly lighter and the urine darker. One week before admission, pruritus appeared. The Van den Bergh reaction showed direct, immediate, and indirect 1.3 units. There was urobilin in the urine. Two attempts to visualize the gallbladder by the Graham-Cole method were unsuccessful.

Gastrointestinal examination on April 4, 1938, showed a delayed motility throughout. Gastric peristalsis was hypoactive. The small intestinal loops were of normal caliber and showed many peristaltic contractions (Fig. 6, *15*).

At operation on April 7, 1938, a calculus was removed from the common duct and the patient made a satisfactory recovery.

It was possible to re-examine this patient on April 12, 1940, and a definite improvement in the gastrointestinal motility was revealed. There was active gastric peristalsis. The small intestinal loops were wider and some contained gas. The mucosal pattern appeared coarser than in the preoperative examination (Fig. 6, *16*, and *17*). The two examinations are compared in Table x and are shown in Chart VI.

CASE 7. N.S., aged fifty-two. A patient of Dr. Ravdin was admitted on July 6, 1939, with a chief complaint of jaundice, itching and loss of appetite. Six months before admission there had been a gradual onset of painless jaundice, malaise, weakness, anorexia, itching, dark urine and clay-colored stools. The Van den Bergh reaction showed, direct positive, and indirect 10 units. Bilirubin three plus in urine. The patient had been taking bile salts orally since March, 1939.

Gastrointestinal examination on July 7, 1939, showed a normal gastric emptying time with

Table XI

Patient N.S.	Stomach Begins to Empty	Stomach Empty	Reaches Colon	Small Intestine Empty	Tone, Pattern, Remarks
Preoperative	Immed.	2½ hr.	Nearly in 5 hr.	c. 7 hr.	Hyperactive gastric peristalsis with commensurate motility. Proximal small intestine dilated with gas-filled loops and motility was slowed. Continuous stream. "Moulage" type of pattern. Colon—increased prominence of mucosal pattern
Postoperative	Immed.	3½ hr.	5½ hr.	c. 7 hr.	Active gastric peristalsis with commensurate motility. Small intestine—loops wider than before, some gas-filled loops

delay in the small intestinal passage time. The proximal small intestine was dilated and showed a coarse mucosal pattern (Fig. 5, *13*).

At operation on July 11, 1939, a calculus was removed from the ampulla of Vater and the patient made an uneventful recovery.

The patient returned for a follow-up examination on April 10, 1940, having been well in the interim, with no jaundice nor acholic stools. The bile salt therapy had been discontinued in September, 1940.

The results of the two examinations are compared in Table XI which follows. The motility was not greatly altered but the caliber of the small intestinal loops was somewhat wider than at the time of the previous examination. There were again gas-filled loops (Fig. 5, *14*).

There was no evidence of gross pancreatic disturbance such as steatorrhea in any of these cases despite the fact that two of the patients were proved to have pancreatic tumors.

Discussion. The normal gastrointestinal timetable allows one and one-half to three hours for the stomach to evacuate the 5 oz.-barium-5 oz.-distilled-water meal.[82] The head of the column should reach the cecum in one and one-half to three hours, and the small intestine should be free of barium in four to six hours.[62]

Judged by these criteria, every one of the patients studied had delayed gastrointestinal motility. The stomach showed hypotonicity and hypoperistalsis in 3 of 7 instances, and in 3 cases, emptying was much delayed.

The small intestinal passage was increased in every case, profoundly so in 5 of the 7 patients. Four patients had gas-filled loops of small intestine; 5 of 7 patients had dilated loops of small intestine, and 6 exhibited a coarsening of the mucosal relief.

From these data obtained from patients who were probably not totally acholic, it is possible to evoke a suggestive, if not pathognomonic, picture of the bile-poor gastrointestinal tract. The stomach empties slowly; the small intestine is dilated, with pockets of gas, and a coarse, flattened mucosal relief particularly in the upper jejunum. There is abundant evidence of peristaltic contractions in the small intestine, yet the meal proceeds so slowly as to give rise to the suspicion of intestinal obstruction.

Summary. The significant findings in the bile-poor gastrointestinal tract are a delayed gastric emptying and a marked delay in the small intestinal passage time. There are changes in the small intestinal pattern which, however, are not pathognomonic of the absence of bile from the gastrointestinal tract.

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DERMATOLOGICAL PROBLEMS IN THE PRACTICE OF RADIOLOGY*

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THE accumulation of ideas and impressions offered in this paper were gained from the many dermatological problems encountered in the general practice of radiotherapy in a fairly small community. The problems often appeared simple, yet many became perplexing and unpredictable in behavior and response. The dissertation will be limited to a brief discussion of a few of the physical aspects of superficial roentgen therapy and a generalization of the treatment of benign skin disorders. The principal theme will develop a reason for withholding aggressive radiation therapy. It is trusted that the younger man starting his practice of radiology or the director of resident training programs will approve the ideas.

The use of roentgen rays or radium should be justified in every case of benign skin disorder. The expected minimal result should overbalance any possible hazard of the accumulated total dose from a schedule of treatment.[5] The skin atrophy and radiation changes from unnecessary treatment may not appear for many years; therefore, one may never see the late sequelae and may not be aware of the changes he produced. The use of roentgen rays for temporary relief should be strongly opposed when it is known that certain skin disorders will recur in spite of repeated small doses of roentgen radiation. For example, recent controlled experiments show evidence that roentgen treatments add very little to the response of acne patients placed on the usual combined routine of irradiation and dermatological care.[6,11] The actual tolerance of the skin to repeated small doses is not known because of the failure to record the time dose relationship in the many reports.[14] However, it seems as though several doses totaling less than 1,000 roentgens in six months is a "safe dose" and above 1,200 r in the same period of time will produce sequelae of "cosmetic significance" (ref. 4—see discussion). The young girl with acne has a skin survival expectancy of fifty to fifty-five years during which time the 1,000–1,200 r will continue its progressive fibrosis in the corium of the skin.[9] Very few patients are warned about the hazard of further treatment elsewhere. In fact, few are told of the agent used.

The possibility of the patient's visiting other doctors for roentgen treatment should be recognized. The drifting patient is a source of concern, and every effort should be made during the interview to "trap the experienced" patient who has suffered a benign recurring recalcitrant itching skin disorder because many of them have made the rounds of the dermatologists and radiologists to find temporary relief from roentgen irradiation, and he will often misconstrue his past history. Everyone is familiar with the dry scale-like telangiectatic skin that eventually forms the typical keratosis. It is amazing to find that ten to twelve treatments had been given by "2 or 3 different doctors."

The dermatologist by virtue of his training is able to recognize the many skin problems at a single glance and understands the underlying pathology. By individual management of the case he may remove or treat the causative agent and produce improvement without actual definitive treatment to the eruption or disorder. Seborrheic dermatitis—acne—allergic eczema are typical examples. He is well aware of the indications or contraindica-

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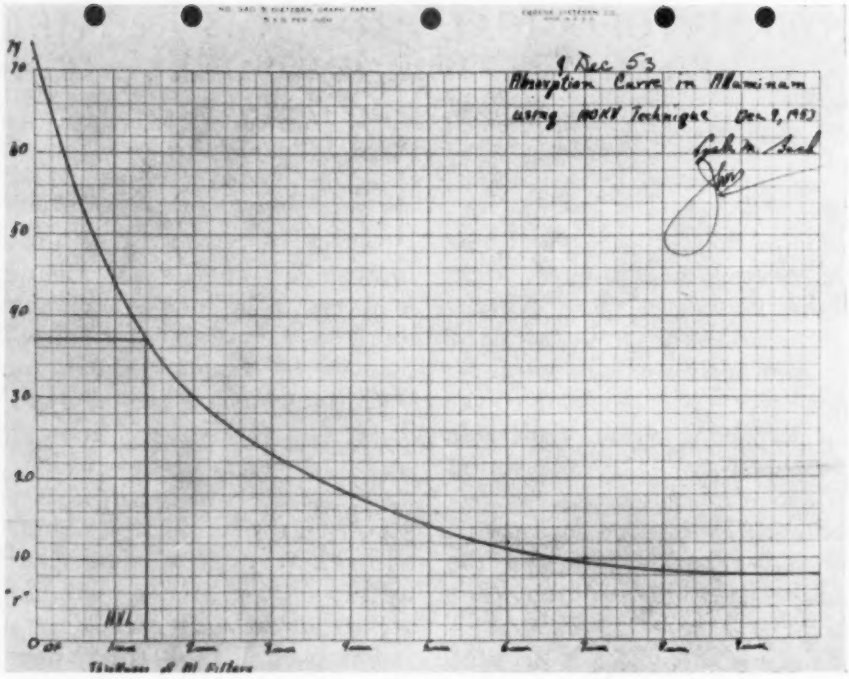

FIG. 1. The half-value layer in aluminum for the low voltages (75–100 kv.) is a necessary part of the calibration of each machine.

tions for roentgen rays or radium. By experience and training he has learned to use roentgen rays as an adjunctive aid to his specialized medicine and topical applications, and often his use of radiation appears empirical.[11] Even though he relies on roentgen radiation as one of the most useful aids, his literature yields very little physical data concerning dosimetry,[10] relationship of size areas, backscatter skin dose or technical beam control, all of which is offered the radiologist in training. By the same token, the resident in radiology would benefit by further training in the recognition and diagnosis of the common skin diseases.

A biopsy will often confirm the diagnosis for the radiologist when he is confronted with an unfamiliar skin lesion.[4] He may then seek therapy information in reference books or literature only to find the dose quoted as "75 r weekly for 6–8 doses." This information leads to "cookbook" therapy which is in many cases undesirable because the directions may be followed to the letter, regardless of the response. Such a predetermined course of treatment should be discontinued at the first or second treatment if there is no response or discontinued if excellent response is obtained by the second or third treatment. In either result, the patient did not receive the best management if definitive dermatological medical care was not carried out.

The primary criticism by the dermatologist is that patients treated by the radiologist receive only roentgen radiation and the etiology and the primary disease are neglected, leading to unnecessary irradiation when medical procedures would control the disorder. It is believed that the radiologist may accept and treat those cases in which the diagnosis is known and in which roentgen irradiation offers control or palliation above any other procedure. Dermatological consultants would clarify the dermatological-medical management.[9] However, in communities where qualified consultants are not obtainable, the radiologist should institute any available means of

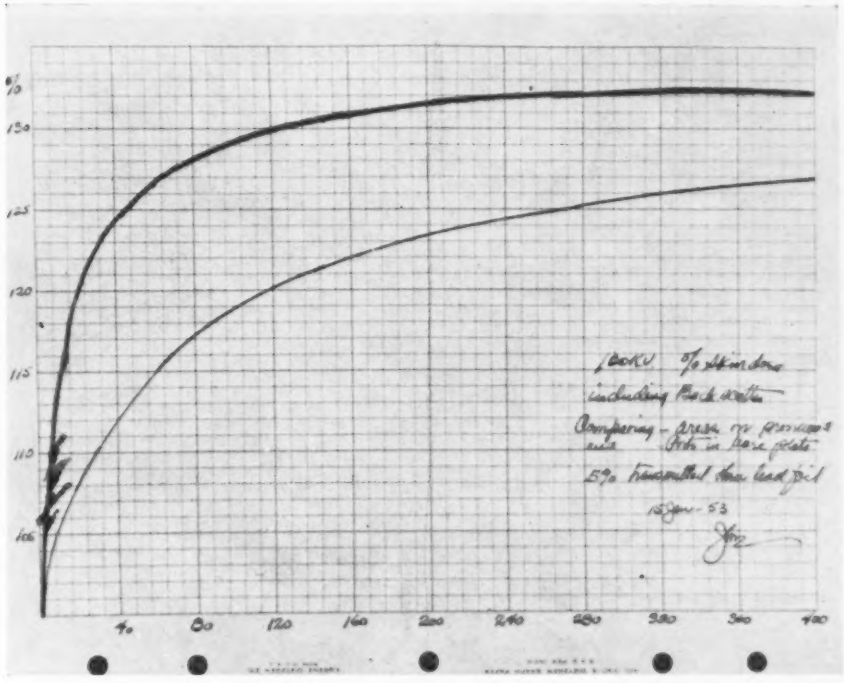

Fig. 2. Skin dose with backscatter is often surprisingly high in large areas used in dermatologic irradiation. The lower curve was plotted from readings in fields formed by various sized apertures in the tube head. The upper darker curve was plotted from readings in various sized fields on the skin (backscatter media) formed by lead foil. The difference amounts to the transmitted and secondary radiation from the foil.

medical treatment supported by roentgen rays or radium in any case in which he feels that the diagnosis is known and that he feels he is qualified to manage in all respects.[11] In this case, he should differentiate benign or malignant lesions and be familiar with those disorders that are amenable to roentgen therapy. Occasionally skin diseases are referred for roentgen treatment of an eruption or skin lesion. Here again, it is believed the radiologist should seek a consultation for the medical care as the primary management and use roentgen therapy as an adjunct. In small cities he may see fit to direct the entire course of events himself including the topical or medical management.

The usual practice in irradiating the skin disease is to deliver a dose of 75 r (air dose) at a specified distance. By comparison, the areas may be large or small and there is a constant laxity toward calculating the skin dose with backscatter. There is the possibility that the response of the skin disease may depend largely on the secondary or backscatter effect. A favorite argument among dermatologists for many years has been the efficacy of the low voltage and "soft x-rays with little penetration." The time intervals between treatments definitely control the immediate response of the disease and tissue reaction[16] and tissue recovery. Remarkably enough, very little is written about volume dose (integral dose) in the 75–100 kv. range in the relatively large areas that are necessarily treated in skin diseases. Perhaps the integral dose within the half-value tissue layer would be useful to define the dose delivered.

Trial measurements to account for the backscatter and skin dose in the 100 kv. range in this department find a shallow curve up to 23 per cent for the areas of 250 cm.^{2} Thereafter, from 250–400 cm.^{2} little increase in the skin dose with backscatter can be measured by the usual methods.

Backscatter readings for the low voltage

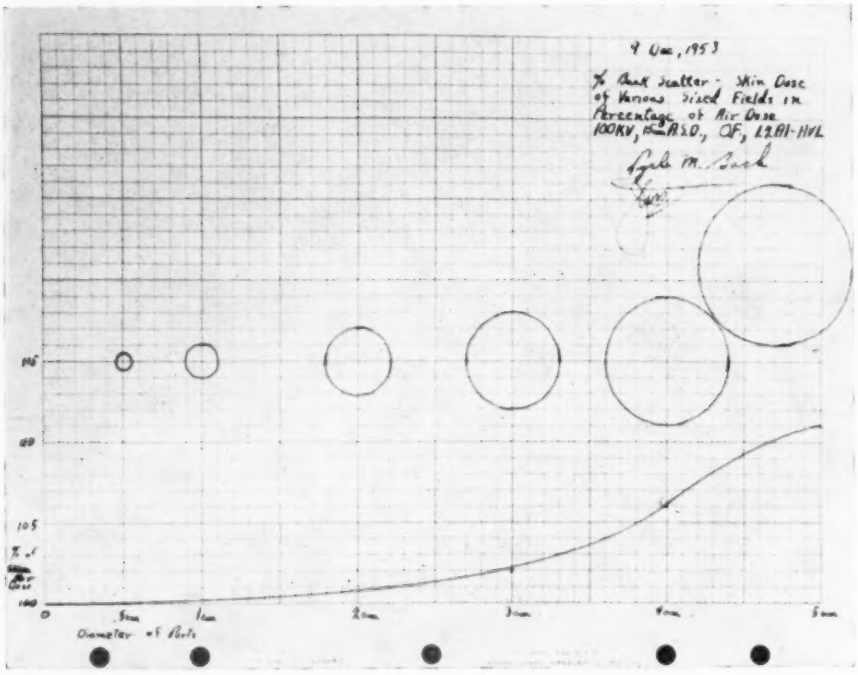

FIG. 3. The skin dose with backscatter is considerably less in the small fields formed by collimating tubular cones. Each should be measured separately.

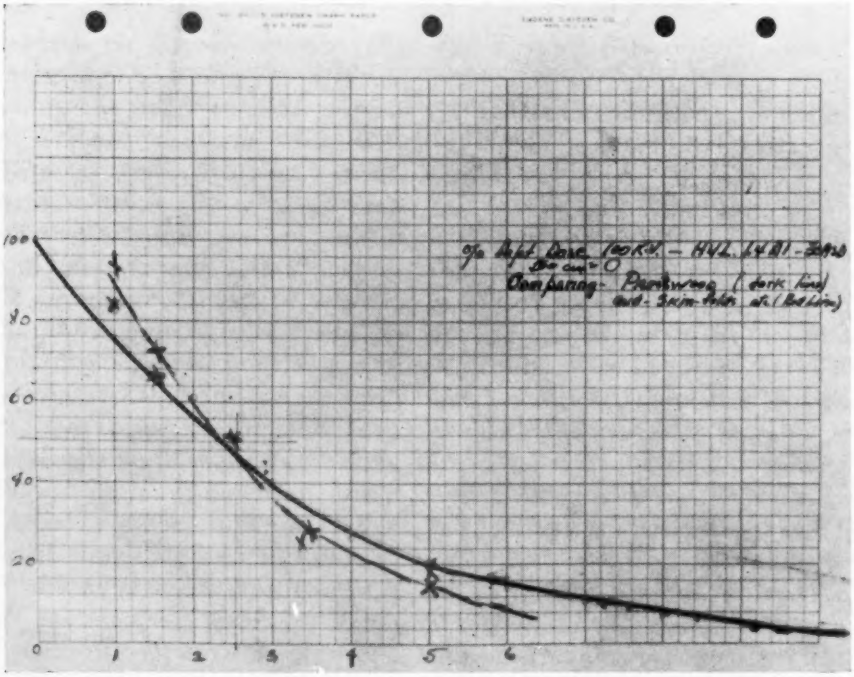

FIG. 4. The absorption curve in presdwood is a useful guide to visualize the depth dose for certain techniques in 100 kv. therapy. The longer curve shows the absorption in presdwood compared with the shorter curve plotted by the "x's," which was the absorption in various thicknesses of fat folds, skin folds and appendages in a patient.

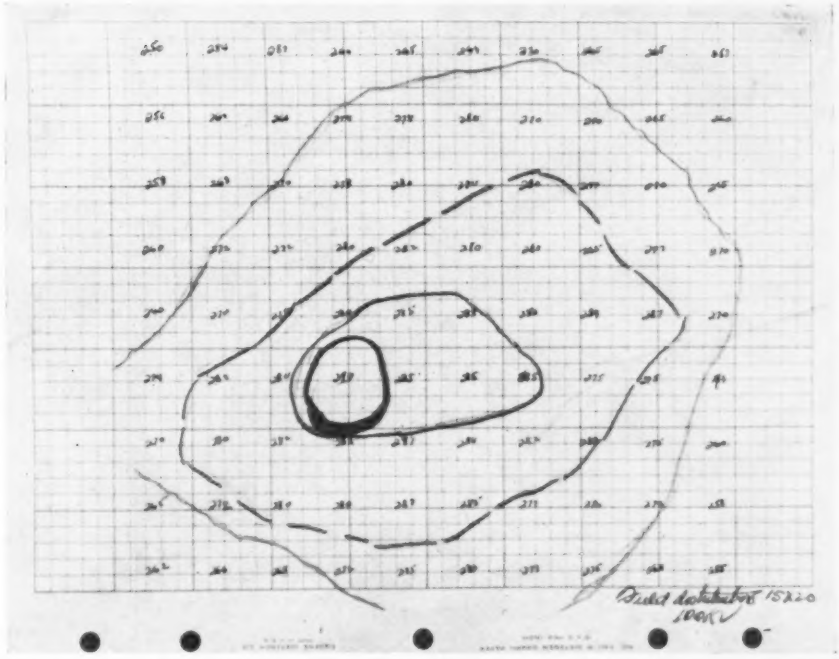

FIG. 5. The field distribution will at times find the central beam eccentrically placed. This study was made with a densitometer and was plotted only for blackening.

(100 kv.) range will vary with the technique of the operator. If the readings are made in fields that are defined by collimating apertures placed in the base of the tube head and compared with readings made in various sized fields defined by lead foil placed directly on the (skin) scatter media, the latter curve will be steeper and will finally level off at points higher than the first curve by a percentage equal to the transmitted and secondary radiation of the lead foil (which is usually 3–5 per cent). Thus, backscatter skin dose curves should be obtained from readings recorded in fields employed by each individual operator and his treatment techniques.

The leveling off of the backscatter dose curve may be considerably influenced by the limiting size of the master cone[7] or the fixed aperture in the tube head. The limiting diameter of the aperture of the tube tank may be small enough to reduce remarkably the estimated size of the large therapy fields. A large roentgen film exposed at various distances will confirm estimated field size that may be obtained through the fixed aperture in the tube tank.

An often neglected exercise is a study of the field distribution from low voltage tubes. The central beam may be eccentrically located. Occasionally a marked "heel effect" is found on the anode side of the beam. Very often the field distribution follows a pattern remarkably similar to the geometry of the reflected (focal spot) cathode image.

There are several disorders in which roentgen therapy is considered to be of little or no value.[9,11] These are listed by the usual clinical terminology. Characteristics and differential diagnosis may be found in a standard text or reference.[1] Among these disorders are acne, lupus erythematosus, actinic keratosis, benign nevi, neurofibromas, hypertrichosis, nevus flammeus (capillary hemangioma skin-port wine stain), xanthomas, and dermatoses of the scalp.[11]

Of the many skin diseases and disorders seen during the past fifteen years of radiotherapy practice, the three most common ones are listed for a short discussion, re-

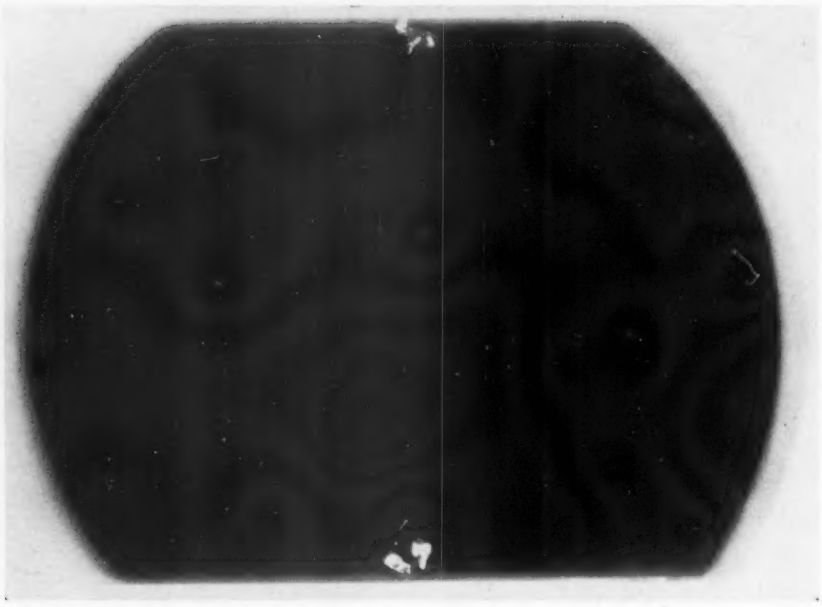

FIG. 6. A large film exposed at treatment distance will confirm the estimated size of the maximum field. All of the graphs and curves shown were constructed by the residents as part of their regular training.

flecting the ideas gained from their management. These lesions are listed for clarity under the usual admitting diagnosis.

1. "*Birthmarks.*"

(a) The plexiform hemangioma (strawberry mark) usually recedes promptly after one or two small exposures of roentgen rays or radium.[12] Spontaneous regression

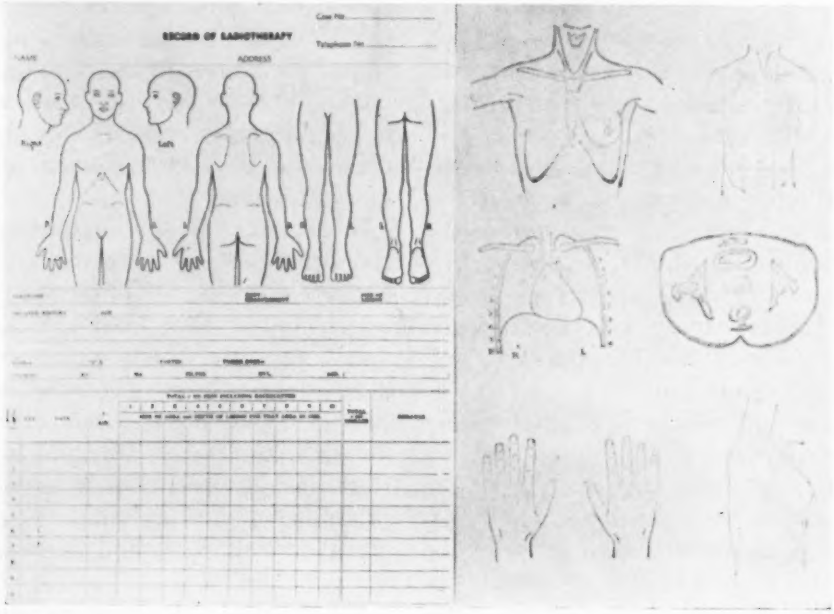

FIG. 7. Accurate charting and recording is essential in any treatment with radiation. Dosage is prescribed in air and calculated for skin with backscatter in varying field size and recorded in an opposite column. Fields are numbered, indicated on a diagram and related to the patient by skin pencil marks or tattoo marks. Anatomical stamps shown in the right half of the illustration are useful.

Fig. 8. The angioma blanches on compression and usually invades the corium. Doses of 100–200 r given at one to three month intervals, totalling 400 to 600 r in nine months will frequently produce recession. The author never exceeds a total of 800 r in nine months.

has been noted after a few years; however, treatment is justified by the rapidly growing lesion and the anxiety of the family. It could never be decided which of the lesions would regress completely and spontaneously; therefore, if growth was confirmed, treatment was instituted. The spontaneous regression reported by several observers could be hastened and the natural growth held in check during the waiting period.

Fig. 9. (No. 7043) A large 1.5 by 3 by 4 cm. bulky hemangioma invading corium, treated with two doses, 200 r (in air) in three months (115 per cent skin dose with backscatter). Five to six seconds of dry ice on two occasions to blanch marginal vessels. Photograph taken three and a half years after last treatment.

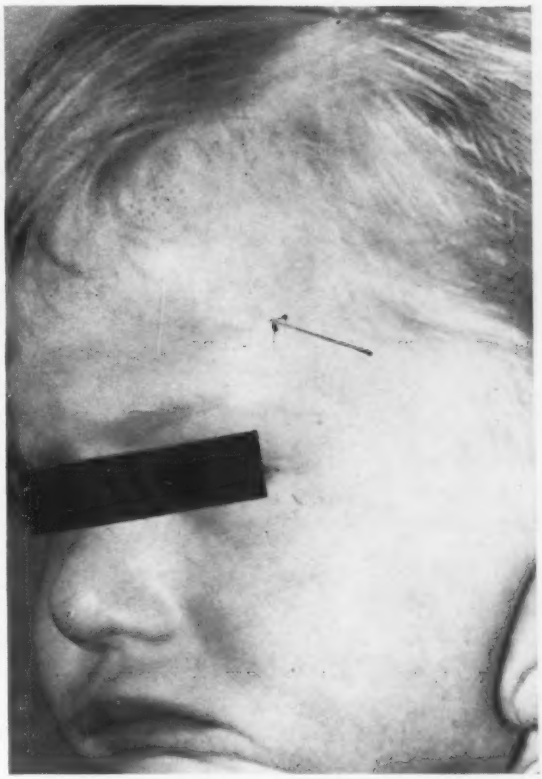

Fig. 10. (No. 9146) A small flat angioma 1 by 1.6 cm. in size treated with three doses, 200 r (in air), 100 kv. in ten months (110 per cent skin dose with backscatter). Dry ice five seconds on two occasions to marginal vessels. Photograph taken three years after last treatment.

Two hundred roentgens (air dose) is the usual initial dose—100 kv., 1–2 mm. Al half-value layer, 15 cm. anode skin distance, 200 r/min. to an area slightly larger than the lesion. Single doses above 300 r are never used in infants.[9] The surrounding skin is screened off by lead foil and a cone slightly larger than the exposed area is used. The dose is repeated in one month if there is no improvement, or the treatment is delayed for an additional three to four weeks if response is marked. A total of 500–600 r (air) may be given in eight months.[9] The usual area will average 3 to 9 cm.^{2} and will accumulate only 105 to 112 per cent skin dose with backscatter. The bulky or tuberous lesions may be compressed by inserting a thin sheet of plastic under the open cone lowered to give

FIG. 11. The bulky, deeper seated hemangiomas, particularly about the face, that persist and show evidence of rapid growth are evaluated for plastic removal. (Courtesy of Dr. John C. Weeter.)

moderate pressure and to increase the depth dose homogeneity. CO_2 snow or the desiccating needle is used to blanch the marginal or residual vessels thus decreasing repeated roentgen dosage and skin reaction.[8] Radon seeds are never used.

Plastic surgical removal is recommended for the angiomas that fail to improve after two treatments (300–400 r in 4 months) or the lesions that are small and located over the joints or flat skull bones.[13] Hemangiomas 1–2 cm. in diameter that appear and grow rapidly after birth and are located in loose, elastic skin folds are referred for surgical removal,[15] particularly if the patient lives at a great distance. Approximately 3 per cent of the newborns have plexiform hemangiomas and about 15 per cent have nuchal capillary hemangiomas (port wine stains).[12]

Only the very small port wine stains (0.3–0.5 cm.^{2}) are treated by electrodesiccation. All others are referred for plastic procedures such as tattooing to skin color or excision. The lesion is very refractory to ordinary means of radiation therapy.[3]

The "traumatic angioma" or the extremely vascular granuloma pyogenicum resulting from a penetrating skin wound is usually avulsed under local anesthesia and the central vessel identified and electrocoagulated.

(b) Moles. The melanomas are all submitted to radical or extended surgery. This lesion is listed because of the importance of recognizing it at any first interview or examination.

Pigmented nevi, soft fleshy moles, brown and hairy moles, linea nevus, fibromas and lipofibromas are all referred for surgical excision.

The moles and nevi are included because of the frequency of patient request for treatment of a "birthmark."

2. *Inflammations.* Patients with acne vulgaris sent to the radiologist for treatment offer the problem of extremely temporary response and immediate recurrence.[2] Recent analysis of the effects of ir-

radiation indicates that little benefit is contributed by roentgen rays.[9] It is believed that roentgen radiation should not be used in the average acne patient except for definite specific reasons of immediate temporary palliative control which is clearly understood by the patient and the doctor.

Boils, carbuncles, cellulitis and erysipelas are usually treated three to four times per week. Varying dosages of 25 to 50 r (air dose) are delivered to an area 1–2 cm. larger in diameter than the swelling—100–140 kv., 1–4 mm. Al half-value layer. Hospitalized patients are usually treated two times a day—total dose of 300 r in one week is never exceeded. The time interval between exposures is increased with improvement. Primary medical, surgical and antibiotic methods are carried out.

3. *Dermatitis and Eczema.* The patient with a dermatitis presents a causative factor, such as the contact agent associated with his work—oils, dust, corrosive materials, lime—any of which should be removed. The fixed and general eruptions produced by medicines and the localized and generalized eczemas offer quite an investigative problem to ascertain and remove the cause. Topical and general medical management is most important. Roentgen irradiation may occasionally be useful to control the fixed eruption after the allergy factor has been eliminated. The usual dose is 75 r (air) to areas no larger than 15 by 15 cm. repeated in five to seven days, with a discontinuance if there is no improvement in two treatments.[8] The total dose does not exceed 300 r (air) in one month. It is useless to pursue the hazard of continued treatment in the face of poor or doubtful response and not to make use of known dermatological procedures and medications that may give a satisfactory result.

SUMMARY

1. Irradiation should be an adjunctive aid in the management of dermatological problems.

2. Its use should be justified by: (a) need for treatment, (b) known response and (c) no other equally effective measure.

3. Minimum dosage should be planned to give maximum benefit and discontinued with acceptable improvement or there is if no immediate response.

4. Careful meticulous records of calculations will serve as guide to future withdrawal or active therapy.

5. More attention should be given to dosimetry and beam control.

6. Departmental radiation hazard should be investigated and avoided.

7. The medical or dermatological management might be the most important part of the treatment and should not be neglected.

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Note of appreciation is offered to Dr. Vincent P. Collins for his patient counsel; to Dr. A. B. Loveman for his many suggestions pertaining to dermatologic conditions and to Dr. Joseph C. Bell for his advice and counsel.

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DISCUSSION

DR. JAMES F. KELLY, Omaha, Nebraska. I think Dr. Love has given us a very nice little sermon this morning on superficial radiation therapy. He certainly is honest. He is honest with himself when he wants an honest diagnosis of what he is treating, and if it doesn't fall in the category of those conditions amenable to roentgen therapy he says, "Give it to the one who can treat it best." That is honesty with the patient, and both angles have to be considered.

There is very little in his paper with which I would disagree. The one thing I might disagree with is the failure to emphasize the treatment of boils on the upper half of the face. I think these boils are essentially for radiation therapy, and I believe they should be treated promptly.

The other item I might question would be the roentgen treatment of angiomas or other lesions over the buttocks in children or around the pelvis. I think a radium plaque with a very low filtration is a much better method of handling these conditions. I don't know of any other way to handle them. We never use any dry ice, but I have seen one case in which harm came to the youngster in whom the dermatologist treated an angioma as one of us might treat an epithelioma.

Dr. Love warns the radiologist against his weakness in diagnosis. He also warns us about the dermatologist whose weakness is in his understanding of radiation factors. The ideal solution is to work together; let the diagnosis come from the dermatologist, and let the treatment be handled by the radiologist.

As far as the other lesions Dr. Love treats are concerned, I don't think any of them are open to any criticism, and there is nothing I can say in the way of emphasizing the value of their radiation management. I might say something about the factors that are used today, and how easy it is for a resident in radiology to approach one of these problems.

Back in 1915, when I started, we had a mercury interruptor and a gas tube, and it was a question of what you had given the patient when you got through. You certainly didn't know. Later on a couple of immortals came along—Clyde Snook with his rectification, and Coolidge with his cathode tube, and then we really had something.

Then the roentgen unit (the r) came in, and people like Dr. Quimby and Dr. Weatherwax came along. That helped us all a lot. From there on it should have been pretty easy, but you still have to use some clinical judgment. Don't forget that. Irradiation will never be what Dr. Love mentioned in his text—this cookbook type of dosage, in which you treat a condition that falls into a certain category with a certain dose. Diseases will never be that consistent in their occurrence, and the patient will never react that consistently to a dose of radiation. You always have to figure the variation in the disease and the variation in the patients' resistance. If you lose track of your clinical attitude in this type of therapy, you might as well go to laying bricks—putting one on top of another. It is easy to do.

When you go back into my group, a lot of us are more apt to have clinical judgment than scientific judgment. Dr. Love puts in the science as well as the practical elements. I want to compliment him on his paper. It is very well written, and should be constructive to all of us.

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Thirty-eighth Annual Meeting: Shamrock Hotel, Houston, Texas, April 9–11, 1956.

EDITORIAL

THYROID-PITUITARY INTERRELATIONSHIP AS DEMONSTRATED BY RADIOIODINE STUDY

THAT close interrelationship existed between the endocrine glands has been known for many years and this became a field for speculation and diagrammatic representation without too much precise knowledge. One of these rich fields for endocrine speculation was the interrelationship between the thyroid and pituitary glands, euphuistically termed the "thyroid-pituitary axis."

With the proven affinity of the thyroid gland for iodine which resulted in a voluminous literature, it was logical to study the extrathyroidal effects in other endocrine glands. It was a natural consequence to see if iodine, either ingested or injected, was deposited in the pituitary gland, and furthermore the close relationship existing between the thyroid gland and the central nervous system led to the study of iodine deposited in the nervous system.

European investigators undertook such studies and while the percentages of iodine deposited in the pituitary gland and central nervous system varied with the investigator there was more or less agreement that the pituitary gland and certain parts of the brain, particularly the interbrain, contained most of the intravenously injected iodine. Schittenhelm[8] explained the differences found by various investigators as due to the fact that the pituitary gland is so small that an analysis must be considered uncertain. He said concerning the titration: "The diminutive weight of the pituitary body results in a proportional increase in the error coefficient."

Schittenhelm and Eisler[8] injected rabbits intravenously for 10 days with a daily dose of thyroxin equal to 1 mg. per kg. of body weight. The thyroxin had a selective action for the interbrain and the latter contained 1,155 gamma of iodine. When 5 mg. per kg. of body weight was given orally in a single dose, within two hours there was 1,000 gamma of iodine in the interbrain—an increase above the normal content of over 100 per cent. Sturm and Schneeberg[9] found after single intravenous injections of thyroid and iodine feeding of rabbits (1 mg. thyroxin per kg. of body weight) values in the tuber cinereum of 260 and 560 gamma of iodine and in the pituitary gland of between 700 and 2,400 gamma of iodine.

According to the analysis of Löhr and Wilmanns[4] the various sections of the brain contain normally between 5 and 35 gamma of iodine. The human pituitary gland contains 60 gamma of iodine according to these authors. This was the average content of 24 pituitary glands. Normally the human interbrain has values ranging from 8 to 21 gamma of iodine.

Following the discovery of various isotopes, it was a natural sequence of events that numerous studies of I^{131} would be applied to thyroid physiology and pathology. Much valuable information has been obtained through this important discovery.

A French group of investigators[2] studied the uptake of radioiodine by the pituitary gland in animals. I^{131} labelled thyroxine injected intravenously into rabbits which were killed two hours after the injection, was found to have concentrated in the posterior lobe. This was not true however, if it was injected in the form of iodide. It was shown that the activity in the posterior lobe of the pituitary-gland was in the form of thyroxin. Concentration of injected iodide did not occur in rabbits in which

thyroxin synthesis had been stopped by antithyroid drugs or by thyroidectomy; but in these rabbits the injection of labelled thyroxin was followed by concentration of radioactivity in the pituitary gland within one hour. The posterior lobe and the tuber cinereum had marked uptake four hours after injection of I^{131} labelled thyroxin, less was found in the infundibulum and only a slight uptake in the anterior lobe and the mammillary bodies.

A. F. Phillips[7] of the Department of Radiotherapeutics at the University of Cambridge had a rare opportunity to study at autopsy a woman aged forty-seven, who died shortly after the administration of radioactive iodine. Eighteen years previously she had a partial thyroidectomy. In 1951 diabetes mellitus was detected and since then she had been on a diet and insulin medication. In June, 1954, she was admitted to the hospital for study. Symptoms of thyrotoxicosis had been present for three months and the blood sugar was persistently elevated. The clinical signs of thyrotoxicosis plus exophthalamos were present. No palpable thyroid remnant was noted. The basal metabolic rate was plus 83 per cent. A tracer dose of radioactive iodine was given which showed a maximum uptake of 80 per cent, and total half-life decay of 4.3 days. Her weight was 59 kg. She collapsed with tachycardia, auricular fibrillation and peripheral circulatory failure. As an emergency measure 2 mc. of radioiodine was given on the second day after her collapse, but she died twenty-three hours later. After death the thyroid gland was found to contain 64 per cent of the administered activity (corrected for radioactive decay). Autopsy revealed pulmonary embolism. The pituitary gland was divided horizontally and the lower half was taken for radioactivity measurements. The weight of the unfixed specimen was 0.43 gm. and the total activity was 0.085 mc. Autoradiographs were made and these showed areas of highly concentrated activity in small groups of cells, some being found on the posterior surface of the gland and others between the pars anterior and pars nervosa. Slight activity was also present at many points in the capsule of the gland and at a few scattered points in pars anterior. The distribution was borne out by the roentgenographic images. Neither pars nervosa nor the colloid-containing follicles of pars intermedia showed autoradiographs above background level.

Phillips in his comment of the findings suggests that the active group of cells in the capsule may not be part of the pituitary gland proper but may be associated with the meninges or vascular system. He calls attention to the fact that the absence of organically bound radioiodine in the pars nervosa is in contrast to the results in rabbits and monkey. He goes on to say that we may be dealing with an abnormal pituitary. In Graves' disease the pituitary mechanism may not only respond abnormally to thyroxin, but also may concentrate it abnormally. I believe this is an important point because there is the possibility that primary exophthalmic goiter has its origin in the hypothalamic-pituitary system. Changes in the pituitary gland have been reported in thyrotoxicosis.[6] Drouet[3] found hyperplasia and increased activity of the anterior lobe of the pituitary gland. He said there are four reasons which show that the role of the pituitary gland in hyperthyroidism is one of hyperactivity: (1) biologic and experimental data; (2) clinical, (3) anatomic-pathologic, and (4) therapeutic considerations. Furthermore antithyroid drugs of the thiouracil type produce changes in the pituitary gland. Several years ago[5] it was stated that "Adults dying as a result of hyperthyroidism have, as a rule, suffered from a severe form of the disease with goiter toxemia. A damaged type of pituitary gland might therefore be found." Such a pituitary gland if in a state of exhaustion would hardly be able to take up iodine. Naturally the uptake of iodine or thyroxin by the pituitary gland would be determined by the state of the cells, because there is such a variation in the degree and length of the

thyrotoxicosis as well as in the age and sex. It must be presumed that final conclusions will require many autopsy studies before the exact interpretation of radioactive findings in the pituitary gland will be evaluated.

Corrigan and Hayden[1] reported a case of a man with prostatic carcinoma with spinal metastases who was treated as a suspected carcinoma of the thyroid. He received I^{131} in small doses for three months and one month before death he was given 5 millicuries. On postmortem examination "There was a trace of activity, not more than 10 per cent above background, in the pituitary but no other indications of activity."

The fact that the hypothalamus and the posterior lobe of the pituitary gland are of nervous tissue origin helps substantiate the argument that the thyroid influences these tissues. It is certain that when more long-term studies are made in individuals who have received radioiodine we will add another chapter to physiologic thyroid-pituitary interrelationship.

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SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS*

NEW GRADUATE PROGRAM IN BIOPHYSICS

A graduate program in Biophysics which leads to either the M.S. or Ph.D. degree has been established by the Sloan-Kettering Division of the Cornell University Medical College. The M.S. program is devoted specifically to Radiation Physics and includes training in clinical radiological physics, medical and research applications of radioisotopes, and health physics. Extensive facilities in the Sloan-Kettering Institute and Memorial Hospital are available for M.S. or Ph.D. thesis problems in various physical, chemical, or biological effects of different qualities of radiation. Related courses can also be scheduled at neighboring Universities. A limited number of fellowships is available.

Additional information on entrance requirements, courses and program details, and fellowship specifications may be obtained by writing: Dr. J. S. Laughlin, Department of Biophysics, 410 East 68 Street, New York 21, New York.

AMERICAN BOARD OF RADIOLOGY

The deadline for filing applications for the Spring 1956 examination is January 1, 1956. Those candidates who will complete their training by June 30, 1956 will be eligible to appear at this examination. For further information, please contact Dr. B. R. Kirklin, Kahler Hotel Building, Rochester, Minnesota.

CANCER FELLOWSHIPS AND RESIDENCIES

Cancer fellowships and residencies are provided for qualified applicants seeking training and research opportunities in oncology at the University of Texas Postgraduate School of Medicine, in the following divisions: diagnostic radiology, therapeutic radiology, anatomical pathology, clinical pathology, experimental pathology, medicine, experimental medicine, surgery, experimental surgery, malignant diseases (rotating), anesthesiology, biochemistry, pediatrics, gynecology, physics, and neurological surgery.

For further information and application forms write to Dr. Grant Taylor, Office of Education, The University of Texas, M. D. Anderson Hospital and Tumor Institute, Texas Medical Center, Houston 25, Texas.

OAK RIDGE RESEARCH PARTICIPATION PROGRAM FOR 1956

Applications are now open to college and university faculty members for places in the Oak Ridge Research Participation Program for 1956.

Administered by the Oak Ridge Institute of Nuclear Studies in cooperation with Oak Ridge National Laboratory, the Research Participation Program is designed to disseminate scientific and technical information to educational institutions by enabling faculty members to spend varying amounts of time—usually the three summer months—conducting research in Oak Ridge laboratories. The participant's Oak Ridge salary approximates his university salary.

Applications for three-month summer appointments in 1956 should be in the hands of the University Relations Division, Oak Ridge Institute of Nuclear Studies, P.O. Box 117, Oak Ridge, Tennessee, by December 15, 1955. Application blanks and further instructions and information may be obtained from the University Relations Division. Notifications of appointment will be made by February 15.

* The list of meetings of Radiological Societies appears in the November, 1955, issue of the JOURNAL, and will be published in the January, 1956, number.

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. LEUCUTIA, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Blood and Lymph System

Schorr, S., Hochmann, A., and Fraenkel, M. Phlebographic study of the swollen arm following radical mastectomy. *J. Fac. Radiologists*, Oct., 1954, *6*, 104–108. (From: Diagnostic and Therapeutic X-Ray Department, Hadassah University Hospital and Medical School, Jerusalem, Israel.)

The swelling of the arm which often follows radical mastectomy is distressing and often disabling. Various causes have been suggested, such as (a) infection of the operative field, with subsequent recurrent lymphangitis and lymphatic obstruction, and (b) venous obstruction. Thirty-five patients were studied in order to evaluate the significance of the venous factor as a cause of swelling of the arm after radical mastectomy. Phlebograms were used to demonstrate and localize the point of obstruction, if any, of a major vein, and to establish whether patency of the cephalic vein alone is sufficient to present edema when the axillary vein is obstructed. Thirty patients with normal arms, undergoing pyelography, were used as controls.

The study showed no correlation between the state of the veins in the axillary region and the swelling of the arm. Apparent venous deformities such as narrowing or defective filling may be due to positioning rather than to disease. Venous obstruction is demonstrated only if sharp narrowing of the vein is accompanied by formation of collateral veins. Of 18 phlebograms performed in patients who had undergone radical mastectomy and had no swelling of the arm, the cephalic vein was demonstrated in 16 cases. The authors think this may prove the importance of the patency of the cephalic vein in preventing postoperative swelling of the arm.—*Samuel G. Henderson, M.D.*

Gvozdanović, V., and Hauptmann, E. Further experience with percutaneous lieno-portal venography. *Acta radiol.*, March, 1955, *43*, 177–200. (From: Roentgen Department and Department of Medicine, University Hospital "Rebro," Zagreb, Yugoslavia.)

The authors review the current literature on this procedure and report a total of 61 cases of their own. This includes the 14 cases presented in their preliminary article 1½ years previously.

The technique used by the authors is as follows: One hour after administration of a sedative, the patient is placed supine on a roentgenographic table. Local anesthesia to the skin is obtained by injection of 1 per cent novocain into the eighth, ninth and tenth intercostal spaces in the anterior axillary line A skin incision precedes the splenic puncture and facilitates the latter. The patient holds his breath on deep inspiration. The needle is pushed 2 to 3 cm. into the spleen and 20 cc. 70 per cent ioduron is injected in two to three seconds. The authors no longer change the cassettes by hand but use an Elema cassette changer, exposing four films per second. Factors used are: 400 ma., 70 to 80 kv. 95 cm. focus skin distance, 0.1 sec., a fine cross grid and a superdynamax tube.

Preliminary intravenous sensitivity test is performed and the patient is made to rest twenty-four hours after the examination. The minor side reactions observed include a sensation of heat and nausea lasting two to five minutes, and pain at the site of puncture lasting twelve to twenty-four hours. The major complications include spread of the contrast medium along the course of the puncture needle, injection into the peritoneal cavity or into the stomach, producing violent nausea and vomiting or into the colon which is asymtomatic. Bleeding from the spleen, both immediate and delayed, constitutes the gravest complication.

Inasmuch as this examination is usually performed on patients with a splenomegaly, a determination of the normal was a prerequisite. The authors present roentgenographic reproductions illustrating the normal position, course and caliber of the splenic and portal veins and their ramifications. The average width of the splenic vein is 1.6 cm. and of the portal vein is 1.9 cm. Anything above this is considered abnormal. The course and position vary somewhat in the normal as well as in various diseases. The splenic vein most frequently crosses the vertebral column at the level of L1, with variations between D12 to L3. The average angle between the splenic vein and the vertebral column is 45 degrees, with variations between 20 to 90 degrees. The speed of circulation was determined by serial films and compared with the normal as established by the authors.

Reflux of the contrast medium into the tributaries of the splenic and portal veins could not be demonstrated in normalcy, but was noted in cases of cirrhosis and thrombosis of the splenic vein, as well as in collateral circulation. Demonstration of the latter depended to a certain extent on the site of the injection of the contrast medium.

The roentgen findings in portal hypertension include definite signs such as a strong reflux into the tributaries of the splenic and portal venous system and the development of collateral circulation, through pre-existing anastomoses, and a marked slowing down of liver circulation. The probable signs include widening of the splenic and portal veins with marked tortuosity.

Two tables and excellent reproductions of roentgenograms of 15 cases accompany this article.—*William H. Shehadi, M.D.*

Kinmonth, J. B., Taylor, G. W., and Harper, R. Kemp. Lymphangiography; a technique for its clinical use in the lower limb. *Brit. M. J.*, April 16, 1955, *1*, 940–942. (Address: Professor Kinmonth, St. Thomas's Hospital, London, England.)

The general principle of the presently described technique is that the lymph trunks are made visible to the naked eye by subcutaneous injection of a diffusible dye so that radiopaque solution can be injected directly into them through a small needle. Lymph vessels, or at least normal ones, are much smaller than arteries or veins used in angiography; they contain colorless lymph and are difficult to see, and under normal conditions may be empty or nearly so, existing as potential spaces like the pleura or peritoneum.

The technical procedure is as follows: (1) The diffusible dye (patent blue violet or patent blue V) is made up into an isotonic solution (11 per cent) in distilled water and sterilized by autoclaving. About 2 to 2.5 cc. of this solution is injected subcutaneously between the toes—about 0.5 cc. into each web, the area being massaged thereafter for about half a minute. (2) The ankle, knee and hip joints are passively moved through full range for about five minutes. The dye is nontoxic but the patient soon becomes blue in color. The dye is excreted in the urine and normal complexion returns in about twenty-four hours. (3) A transverse incision (in a bloodless field) is made 2 inches proximal to the webs of the toes and the vessels, which lie very close to the skin and are about 1 mm. in diameter normally, are visualized filled with blue dye. Dissection must be very cautious. Pressure above the incision and massage below it help to distend the vessel. (4) The radiopaque solution injected is 70 per cent diodone, a No. 18 hypodermic needle on a 10 cc. syringe being utilized. The vessel becomes white and transparent if injection is accurate; otherwise a small bleb forms at the site. The full 10 cc. is injected as rapidly as the bore of the needle will allow. Good delineation has been obtained with as little as 1 cc. of injected material. (5) Filming is done as soon as possible, the normal vessel appearing as a fine opaque filament in the soft issues. Films in holders or cassettes are placed end to end under the entire extremity and a special type of tube arm permits filming of the whole length of a limb. A wedge filter compensates for varying extremity thicknesses. For distal extremities, nonscreen films, factors of 65 kv., 65 ma., 2 seconds at a 48 inch distance or for thicker parts, with grid cassettes, 80 kv., 50 ma., 1½ seconds, at the same distance have been used.

The method has been employed in lymphedema praecox, postmastectomy edema of the arm, postphlebitic ulceration and edema of the leg and other diseases. The lymph vessels normally remain the same width in their course up the limb, unlike veins which become wider. Ampullae of valves are often seen.

Reproductions of normal and abnormal lymphangiograms are included.—*Chauncey N. Borman, M.D.*

General

Williams, Ifor. Calcification in loiasis. *J. Fac. Radiologists*, Oct., 1954, *6*, 142–144. (From: Department of Diagnostic Radiology, University College Hospital and the Hospital for Tropical Diseases, London, England.)

Loiasis is a relatively benign disease, endemic in many parts of West Africa. It is caused by filarial parasites in the larval state, occurring in mosquitoes and flies, being transmitted to man. Here they develop into adult worms, inhabiting the tissue spaces. The salient features are the presence of adult worms beneath the skin, especially near the eye, and Calabar swellings. The clinical manifestations may be months or years in developing, and then persist for ten years or more in the absence of effective treatment. The diagnosis is based on the possibility of infection, a story of adult worms or Calabar swellings, and intradermal and complement fixation tests.

The roentgenological appearances are distinctive. Thin white threads are seen against the soft tissue shadows lying coiled or angulated in the subcutaneous layers. They may be up to 4 cm. in length and less than 1 mm. in width. The thread-like shadow may be fragmented, and tiny rods and dots of calcium may be the residuum. The tendon sheaths may be thickened and visible as soft tissue shadows.

Case histories of 3 patients with loiasis showing calcifications in the hands, compatible with calcification of dead adult worms, are recorded.—*Samuel G. Henderson, M.D.*

Kahn, Alfred, Jr., and Fulmer, John. Acrocephalosyndactylism. *New England J. Med.*, March 10, 1955, *252*, 379–382. (From: Departments of Medicine and Ophthalmology, University of Arkansas School of Medicine, Little Rock, Arkansas.)

A case of Apert's syndrome or acrocephalosyndactylism is presented with a comment on the history of the disease.

There were 48 published and verified cases up to 1932. The authors pursued studies from 1932 to date, and the conclusion was "that there has been no new or important discovery regarding causation and therapy."

The congenital syndrome usually consists of fusion of the digits and a high head, highest point being the bregma. Individual cases may reveal minor accessory abnormalities.

Park and Powers observed "that there is bone involvement as well as soft tissue; the more distal parts are most maldeveloped."

The malformations are bilateral, symmetrical; the malformations are not new formations.

The etiology considered has been defect in germ plasm, fetal infection, injury to nervous system, pituitary disorder, tuberculosis, toxoplasmosis, etc.

A case is presented which is described as fully investigated. The results suggested endocrinopathy: the sella turcica was eroded and there was a positive Keffler-Power-Robinson Water test for adrenal insufficiency.

The blood sugar was above normal.

Continuing effort is recommended to help keep these patients adjusted to their environments.—*Wallace M. Roy, M.D.*

Weiss, Howard, and Crosett, Alexander D., Jr. Chondroectodermal dysplasia. *J. Pediat.*, March, 1955, *46*, 268–275. (From: Hunter Air Force Base Hospital, Savannah, Ga. Present address, Dr. Weiss, 55 E. Washington St., Chicago, Ill.)

A sixth case of chondroectodermal dysplasia (Ellis-van Creveld syndrome) is reported. The diagnosis was made on the second day of life.

The infant, a male, presented the characteristic tetrad of chondrodysplasia, ectodermal dysplasia, polydactyly and a congenital heart malformation. As in previously reported cases, the chondrodysplasia has been manifest by relative and absolute shortening of the extremities with thickening and progressive distalward shortening of the tubular bones, the fibula being most shortened of all. Dysplasia and hypoplasia of the acetabula were also evident in this patient. Tiny, deformed fingernails, malformed teeth and fine, scanty hair were the characteristic features of the ectodermal dysplasia and, in addition, this infant had an epispadias. The polydactyly was accompanied by syndactyly and synmetacarpalism. The congenital heart malformation was probably an interventricular septal defect.

At nine months of age, after several upper respiratory tract infections and episodes suggesting hepatitis, this infant is doing well, is alert and apparently of normal intelligence.

The prognosis appears to depend on the severity of the malformations, particularly of the heart. Of the reported cases, no mortality has been recorded.—*H. G. Peterson, Jr., M.D.*

RADIATION THERAPY

Kohn, Henry I. Changes in the human leukocyte count during x-ray therapy for cancer and their dependence upon the integral dose. *Radiology*, March, 1955, *64*, 382–391. (Address: Radiological Laboratory, University of California Medical Center, San Francisco 22, Calif.)

Forty-eight selected patients undergoing a conventional course of roentgen therapy, planned for four to seven weeks, were followed in order to determine the change in count of each kind of leukocyte. The tumor dose varied from about 3,000 to 6,000 roentgens and the integral dose varied from about 1 to 50 megagram-roentgens. The estimation of the integral dose can be obtained from a table which is presented. A new term, specific integral dose, is introduced. This is defined as the integral dose per kilogram of body weight.

Multiple blood counts done under as near standard conditions as possible prior to, during, and following roentgen therapy showed no significant change related to radiation dose for the basophil, eosinophil, or monocyte counts, or for the neutrophil counts in patients receiving less than 10 megagram-r. In patients receiving more than 10 megagram-r, the neutrophil count, though variable, on the average fell to about 70 per cent of its initial level by the middle of the treatment period, thereafter remaining at about that level.

Throughout the course of treatment, the lymphocyte count tended to fall gradually. For irradiation of the trunk, neck and pharynx, the magnitude and rate of fall of the lymphocyte count depended primarily upon the integral dose and integral dose rate rather than upon the tumor dose or the kind of tissue irradiated. Several exceptions to this were noted, however. At integral dose rates of 0.25 to 1 megagram-r per day, the smaller the daily dose, the smaller the total integral dose required to reduce the lymphocyte count to 25 per cent of its initial level.—*Donald N. Dysart, M.D.*

Allen, Kenneth D. A., and Freed, John H. Correlation of field size and cancerocidal dose in x-ray treatment of skin cancer. *J.A.M.A.*, April 9, 1955, *157*, 1271–1274. (Address: Dr. Allen, 227 16th St., Denver, Colo.)

In the literature concerning the treatment of skin cancer insufficient emphasis has been given to the importance of dosage in relation to the size of the field irradiated. For the purpose of evaluating this factor the authors analyzed 1,013 cases of primary cancer of the skin and lip, treated by roentgen therapy from 1930 to 1953. Five hundred and sixty-four of these followed one year or more were used as the basis of this study. Lesions up to 1 square centimeter in size were treated with 4,300 to 4,700 roentgens in two days. Those from 1 to 6 square centimeters were given 3,200 r in five days. Large, thick lesions were treated over a period of ten to twelve days. Ninety to 130 kv. was used for the smaller lesions and 250 kv. for the large, thick lesions.

When the cases were plotted on a double logarithmic paper with tumor dose as the ordinate and time in days as the abscissa, the slope of the resulting curve corresponded to that determined by Strandqvist previously. The latitude of safe dosage was

found to increase markedly with fractionation and to decrease with increase of size of field. In the former case the range of safe dosage increased from 1,750 r for a single massive dose to 2,800 r if fractionated over twelve days, using a field under 2 square centimeters. With a field over 9 square centimeters, however, the range of safe dosage decreased to 500 r for a single dose and 1,200 r for the fractionated dose.

In the group of 564 cases there were 18 recurrences. All of these were at the margins of the lesions and apparently due to the use of too small a field rather than to underdosage. There were 24 cases of mild radiodermatitis (telangiectasis) and 8 cases of severe radiodermatitis (delayed healing or mild late necrosis). Treatment must be individualized according to the condition of the skin surrounding the lesion, the location of the lesion and the multiplicity of neighboring lesions. Skin with relatively poor collateral blood supply such as on the back of the hand, the leg or the back and skin immediately overlying cartilage tolerates radiation poorly.—*Leslie K. Sycamore, M.D.*

Horrax, Gilbert, Smedal, Magnus I., Trump, John G., Granke, Richard C., and Wright, K. A. Present-day treatment of pituitary adenomas; surgery versus x-ray therapy. *New England J. Med.*, March 31 1955, *252*, 524–526. (From: Departments of Neurosurgery and Radiology, Lahey Clinic, Boston, Mass., and High Voltage Research Laboratory, Massachusetts Institute of Technology, Cambridge, Mass.)

The material and statistics in this article represent a careful survey of all adenomas of two main types at the Lahey Clinic.

From November 1, 1932, through December 31, 1953, 303 patients were admitted to the Lahey Clinic with the diagnosis of either a chromophobe or a chromophile pituitary adenoma. In 143 patients this diagnosis was verified histologically by material removed at operation.

In the other 160 cases the diagnosis was based on the demonstration of a large sella turcica by roentgenography, combined with defects in the visual fields, optic nerve pallor or outspoken evidence of pituitary disturbance. Of the total 303 patients adenoma of the chromophobe type was diagnosed in 243, and the other 60 gave acromegalic evidence of chromophile adenoma.

The authors are interested in comparing the results of treatment during two periods; the first, before use of the 2,000,000 volt apparatus at the Massachusetts Institute of Technology, and the second, employing rotational therapy in all pituitary adenomas.

For this purpose the patients were divided into two groups: those seen and treated from 1932 to 1949 inclusive, and those seen from 1950 to 1953 inclusive.

In the period 1932 to 1949 inclusive a total of 125 patients were treated by surgery only, or by surgery combined with irradiation. Of this number, 108 survived the primary operation, and 79, or 73.1 per cent, were improved for periods varying up to eighteen years, with several unheard from, so that this figure may be somewhat low.

In this period 173 patients were treated by irradiation only or were operated on because they had received no benefit from the irradiation. However, of this total number 74 patients showed no visual loss or other serious symptoms such as intractable headache in acromegaly.

Therefore, 99 patients were treated for visual loss, or for severe headache and progressing acromegaly. Of this number 58, or 58.5 per cent, had to be operated upon since radiation therapy with the usual type of apparatus was not effective.

The radiation given during this earlier period consisted of a total dose of 1,800 to 2,400 r delivered to the tumor through two temporal portals in most cases. If vision was not improved or had not deteriorated seriously after two or three months, a similar series of radiation treatment was given. In some cases a third or fourth series was used. However, if vision deteriorated or was not held at a useful level within three to six months, operation was advised.

In the recent four-year period, 1950 to 1953 inclusive, a total of 63 patients of both types was seen and treated. Ten of these were operated upon without having had irradiation. There were 8 patients in whom vision was so poor (usually 20/200 or less) that operation was considered imperative before a trial of irradiation.

There were 53 patients who received radiation therapy with the 2,000,000 volt apparatus at the Massachusetts Institute of Technology, all of whom received a tumor dose of 4,000 r by the rotational method. Fifteen of these patients were treated for glandular difficulties only and showed no visual loss or other serious symptoms, leaving 38 patients who had failing vision for which treatment was instituted. At varying intervals after treatment it was considered necessary to operate on 7 patients because vision did not improve, this number representing 18.4 per cent of the 38 patients with visual loss. A comparison of this figure with the 58.5 per cent of patients who had to be operated on in the period before rotational therapy shows the gain afforded by this method of treatment.

Although the length of time since the treatment only runs up to four years, 30 of the 38 patients with visual loss or serious symptoms of other kinds have shown excellent improvement for eight months to four years, with 4 patients stationary and 4 others either too recent or not heard from.

On review of the present material, it seems almost

certain that considerably larger amounts of radiation should have been given to the patients treated before the use of the 2,000,000 volt apparatus, even though they might have been subjected to permanent hair loss. Present day results are apparently better due to the larger dosage.—*Stephen N. Tager, M.D.*

McWhirter, R. Simple mastectomy and radiotherapy in the treatment of breast cancer. *Brit. J. Radiol.*, March, 1955, *28*, 128–39. (From: Department of Radiotherapy, Royal Infirmary, Edinburgh, Scotland.)

There has been considerable controversy regarding the value of radiation therapy as an adjuvant to surgery in the treatment of breast cancer. The only realistic survival rates are those based on all cases referred to a general hospital and not just a fraction of the total cases selected according to strict criteria of operability. These more realistic survival rates reveal that radical mastectomy alone fails in a high proportion of cases.

A total of 1,882 cases of carcinoma of the breast was analyzed from 1941 to 1947. The five and ten year survival rates were determined. All but 252 cases had histological confirmation and these were so far advanced that no surgery was done. The cases were placed into three groups according to a slight modification of the Manchester classification of 1946. The operable group included the Manchester Stages 1 and 2. The locally advanced group included Stage 3 and approximately one-half of Stage 4. The distant metastatic group included the remainder of Stage 4. The operable group comprised 56 per cent of the cases, the locally advanced group 29 per cent, and the distant metastatic group 15 per cent.

The main method of treatment was simple mastectomy and a full course of radiation therapy. Radiation therapy consisted of delivering a minimum tumor dose of 3,750 r in a period of three weeks to the chest wall and the whole length of the chain of axillary lymph nodes, including the supraclavicular lymph nodes. The internal mammary lymph nodes were not always fully included at first, but later they were included. The maximum dose at any point of the tumor was fixed at 4,250 r. The half-value layer of the roentgen rays used was 3.7 mm. of copper.

Breast cancer may spread to the axillary, internal mammary, supraclavicular, and posterior cervical groups of lymph nodes. The internal mammary lymph nodes are involved in 48 per cent and the supraclavicular lymph nodes in 33 per cent of the cases when the axillary lymph nodes are found to be positive. Wangensteen (1952) showed that when the axillary lymph nodes were positive, either the supraclavicular or the internal mammary group of lymph nodes was involved in 60 per cent of the cases.

The crude five year survival rate showed 58 per cent of the operable group living, 30 per cent of the locally advanced group, and 4 per cent of those with distant metastases. The crude ten year survival rate showed 39 per cent of the operable group living, 15 per cent of the locally advanced group, and none with distant metastases. Pregnancy or lactation in cancer patients without distant metastases showed a five year survival rate of 40 per cent in the operable group and 27 per cent in the locally advanced group. These survival rates are significantly higher than those previously obtained by radical mastectomy and postoperative radiation therapy.

Twenty-two per cent of the total cases had no axillary lymph node involvement and 34 per cent had axillary involvement, 20 per cent of which also had supraclavicular or internal mammary lymph node involvement. Radical mastectomy would therefore be of value only in 14 per cent of the total cases. This 14 per cent is called the "disputed group."

Handley (1952), employing radical mastectomy in 16 cases in the "disputed group," reported 10 survivors at the end of thirty-three months. It was surmised that at the end of five years the survival rate would be 50 per cent. The survivors would thus represent only 7 per cent of the total cases. If radiation therapy were added in this group to simple mastectomy, the results should be as effective as radical surgery.

Radical mastectomy can be usefully employed only in 36 per cent of the total cases. Simple mastectomy and radiation therapy can be extended to 85 per cent of the total cases and thus offer a cure to many more patients.

A satisfactory radiation therapy service is essential before this method is more widely adopted.—*Charles W. Cooley, M.D.*

Heyman, James. Some problems of current interest relating to classification and treatment of uterine carcinoma. *Am. J. Obst. & Gynec.*, March, 1955, *69*, 502–509. (From: Department of Gynecology, Radiumhemmet, Stockholm, Sweden.)

As editor of the nine volumes of "The Annual Report on the Results of Treatment in Carcinoma of the Uterus," Dr. Heyman discusses several problems related to staging and presents a few of his own statistics.

The usual four stages in carcinoma of the cervix apparently have been almost universally accepted. It is recommended that Stage 0 (pre-invasive carcinoma) continue to be listed separately, since the reported cases are too few to be statistically significant as far as results are concerned.

If the results of surgery in Stage 1 cases are to be compared with those of radiation therapy, surgical cases must be staged prior to operation and that staging maintained regardless of subsequent findings. If such a procedure is followed with the cases re-

ported in Volume 9 (1954) of "The Annual Report" it would appear that the five year recovery rate in Stage I carcinoma of the cervix at institutions favoring primary surgery is 62.5 per cent, compared with 65.3 per cent at those favoring primary radiation therapy. As yet, the value of primary surgery in Stage I carcinoma is not proved and it is desirable that the responsibility for the establishment of such evidence should be left to only the most expert gynecological surgeons.

Classification of carcinoma of the corpus reserves Stages I for disease confined to the uterus and Stage II for disease extending beyond the uterus. Heyman recommends subdividing Stage I into: Group 1 to include cases in which operation is advisable and Group 2 to indicate cases which cannot be operated because of some complicating factor other than spread of disease. He admits the latter is subject to varying opinion as to operability. "Microscopically debatable" cases of corpus carcinoma are classified as Stage 0 in "The Annual Report."

The author treats carcinoma of the corpus by primary intracavitary radium with surgery reserved for treatment failures, although he states that he has never claimed superiority of radiation therapy over the combined methods of surgery and irradiation. He does not agree that hysterectomy is the therapy of choice but that the methods used should be those for which best facilities are available. Further experience is necessary before one method can be shown to be superior to another.

To cover the cases in which the origin of presumably uterine carcinoma cannot be determined, four additional groups are listed in "The Annual Report": carcinoma vaginae et cervicis, carcinoma corporis et endocervicis, carcinoma uteri et ovarii, and carinoma pelvis.—*Henry J. Klos, M.D.*

Beecham, Clayton T., Peale, Augustin R., and Robbins, Robert. Nitrogen mustard and x-ray in the treatment of pulmonary metastases from choriocarcinoma. *Am. J. Obst. & Gynec.*, March, 1955, *69*, 510–524. (From: Departments of Obstetrics and Gynecology, Pathology, and Radiology, Temple University Hospital and Medical School, Philadelphia, Pa.)

Of 74 authentic cases of choriocarcinoma recorded by the Chorionepithelioma Registry of the American Association of Obstetricians and Gynecologists in 1954, 41 had pulmonary metastases, 2 of whom were alive six months after therapy. One of these cases was treated by the authors and at the time of this writing she was alive twenty-seven months after therapy. This, and another case similarly treated, form the basis for the present report.

The more favorable result was obtained in a twenty-two year old white female who had a panhysterectomy for a proved chorioepithelioma of the corpus, even though she had bilateral pulmonary metastases. She received testosterone, 100 mg. per day, beginning twelve days postoperatively. Four weeks later her condition was deteriorating, with increase in size and number of the pulmonary metastases. At this time, roentgen therapy was begun to the lungs, giving 2,100 r to the mid plane of each lung in three weeks (half-value layer of 1.7 mm. Cu, 9 by 20 cm. fields, 2 anterior and 2 posterior, 2 fields treated per day). About two-thirds of the way through the roentgen therapy, 20 mg. of nitrogen mustard (schedule not given) was administered.

Roentgenograms three weeks after therapy showed regression of the pulmonary lesions and three months later the lungs appeared normal and remained so. Clinically she also became well, the hormonal studies indicating cure of the disease.

The second case was similarly treated. She, too, had panhysterectomy but also removal of a vaginal metastasis. Her pulmonary symptoms were severe and she improved markedly following irradiation of the lungs and nitrogen mustard therapy. However, she developed pelvic recurrence and died three months after operation.—*Henry J. Klos, M.D.*

Emmett, John L., and Winterringer, James R. Experience with implantation of radon seeds for bladder tumors: Comparison of results with other forms of treatment. *J. Urol.*, March, 1955, *73*, 502–515. (From: Mayo Clinic and Mayo Foundation, Rochester, Minn.)

This article is a review of the reported experiences of the treatment of bladder tumors. In summary, the authors indicate that the five-year survivals of all patients with bladder carcinoma can be expected to be approximately 33 per cent. The degree of infiltration of the tumor is probably the most important single factor in prognosis, although the size of the tumor, its location and grade of malignancy, the presence or absence of urethral obstruction and the presence or absence of metastasis are also important.

Statistics at the Mayo Clinic indicate that radon seeds had been used in only 5 per cent of patients in the past. This treatment had been reserved chiefly for palliation for inoperable tumors or for those patients in whom the risk of open operation was excessive and where the size and location of the tumor interfered with transurethral removal. A study of 118 patients in whom radon seeds were implanted for bladder carcinoma shows that 29 per cent of the 115 who were traced survived five years or more. In the large majority of cases, recurrences or continued uncontrolled tumor were apparent within the first two years and most of these within the first year. A patient surviving two years without recurrence has an excellent chance of surviving five years or more. Postirradiation cystitis was not a troublesome complication in this group of patients.

The authors believe that as a result of this study the combined treatment of transurethral removal plus implantation of the radon seeds may merit more frequent use in the future.—*George W. Chamberlin, M.D.*

RADIOISOTOPES

Meschan, I., Oddie, T. H., and Regnier, George, with the technical assistance of Young, Clifton C., and Jackson, Calvin C. A method of dosimetry for carcinoma of the cervix utilizing a modified Manchester technic with cobalt 60. *Radiology*, April, 1955, *64*, 546–559. (From: Department of Radiology, University of Arkansas School of Medicine, Little Rock, Ark.)

A method of dosimetry for carcinoma of the cervix, using a modified Manchester technique with cobalt 60, is described. It involves the use of a new applicator, claimed to have all the advantages of the Manchester system, with the added advantage of a rigid relationship between the tandem and the colpostat sources.

Certain anatomical fixed "points" are defined, towards which treatment is directed. Dosage delivered to these points, using the roentgen as the unit of dosage, is readily calculated. The points in the pelvis which they tabulated and used for reference are as follows:

(1) Point C, 0.7 cm. lateral to the center of the external os of the cervix.
(2) Point C^1, 0.7 cm. superior to the center of the external os of the cervix.
(3) Point A, 2.0 cm. superior to the level of the lateral fornix and 2 cm. lateral to the central uterine axis.
(4) Point W, indicating the lateral wall of the pelvis.
(5) Point Bl, on the posterior wall of the base of the bladder.
(6) Point R, the point on the rectum nearest to the intracavitary applicator.

A summary of the method of treatment employed in each of the four stages of spread of carcinoma of the cervix is included. A short method of calculation of dosage is applicable when it is possible to make a symmetrical application. When this is not possible, a more complicated method of calculation is required.

Charts, tables, and isodose curves are reproduced. —*A. W. Sommer, M.D.*

Oddie, T. H., and Meschan, I. The physical aspects of the utilization of the tripartite rigid cobalt 60 applicator for the treatment of carcinoma of the cervix. *Radiology*, April, 1955, *64*, 560–566. (From: Department of Radiology, University of Arkansas School of Medicine, Little Rock, Ark.)

The authors give in detail the basic physical considerations in the use of their cobalt 60 applicator for the treatment of carcinoma of the cervix. By means of their special dosimetry tables and roentgenograms corrected for magnification, it is possible to obtain the gamma roentgen contribution to any point in the pelvis. By predicating certain fixed relationships between the rigid applicator and these pelvic points, an ideal distribution of radioactivity in each capsule of this applicator has been determined.

When a symmetrical application is obtained, a short method of calculation of dosage may be used. If a symmetrical application cannot be done a longer method of dosage calculation is required.

Tables are included giving the calculated individual dosage rates at various pelvic points for 1 mc cobalt 60 in each source of the applicator. Similar tables give the combined dosage rates.—*A. W. Sommer, M.D.*

Hinman, Frank, Jr., Schulte, John W., and Low-Beer, B. V. A. Further experience with intracavitary radiocobalt for bladder tumors. *J. Urol.*, Feb., 1955, *73*, 285–291. (From: University of California Hospital, San Francisco 22, Calif.)

The authors indicate that adequate carcinocidal dosage for bladder tumors may be in the range of 7,000 to 8,000 tissue r in a period of fifty to sixty days. In those instances where the patient's condition or the stage of disease makes the possibility of complete eradication unlikely, a dosage of a palliative value is given. This may amount to 3,000 to 4,500 r in a period of forty days. In other patients a palliative beneficial effect of temporary nature may be obtained with doses varying from 300 to 1,500 tissue r.

Radiocobalt is used because it offers nearly a point source which can be placed in a geometrical center of the bladder so that homogeneous irradiation covers the mucosal surface. In this article 34 patients are reviewed. Ten patients had lesions which were confined to the mucosa or penetrating less than half the thickness of the muscle, 22 patients had infiltrating tumors classified as penetrating more than half the thickness of the muscle or extending beyond the bladder wall.

Treatment with radiocobalt was given in two sittings at seven to ten day intervals. The dosage at each setting was approximately 2,500 r to 3,000 r as measured at the mucosal surface. The average dose of external irradiation with roentgen rays was around 3,500 r in thirty-five to forty days. These had the quality of 0.95 mm. copper half-value layer or 2.5 mm. copper half-value layer.

Nine patients with noninfiltrating tumors had adequate irradiation. Of these 9 patients, 4 are without evidence of disease up to four years from the time of initial treatment. Two patients developed new lesions which were subsequently treated by trans-

urethral resection. Three patients had persistent lesions and 1 died of cancer. Of the infiltrating group of lesions, 14 of 22 had what was considered adequate radiation therapy. Ten of these patients died of cancer and 3 are arrested at the time of the writing of this article. One patient has persistent cancer present in the bladder.

In most patients who had adequate radiation therapy, there is a relatively high incidence of radiation cystitis, contracted bladder and gangrenous cystitis.

Irradiation by the cobalt 60 source in the bladder seems to be indicated for those patients who have multiple bladder lesions, exceeding one to three in number. The severity of the postirradiation complications is in large part dependent upon the fractionation of the dosage and the over-all time factor. Infiltrating lesions cannot be treated by intracavitary cobalt alone but may be treated by intracavitary cobalt and external roentgen therapy in some patients; however, other measures are often necessary because most of the infiltrating lesions are only temporarily arrested by the combined intracavitary and external radiation therapy.—*George W. Chamberlin, M.D.*

VERMOOTEN, VINCENT. Use of radioactive cobalt (Co_{60}) in nylon sutures in treatment of carcinoma of bladder: preliminary report. *J. Urol.*, Feb., 1955, *73*, 280–284. (Address: 2609 Welborn St., Dallas 4, Texas.)

This article includes 2 case reports in which a special technique using radioactive cobalt nylon threads as sutures in a bladder carcinoma and in a secondary carcinoma of the bladder from a primary lesion in the prostate was employed.

The bladder was exposed suprapubically and mobilized so that the nylon threads could be sutured through the tumor from the external bladder surface. The lesion in one patient was a transitional cell carcinoma invading almost completely through the muscularis. Unfortunately, no dosage factors are given. The nylon cobalt threads were left in place for six days and were then removed through the suprapubic opening. The patient had a good result, although a secondary operation and insertion of the cobalt sutures was necessary four months later for a persistent tumor in the bladder base.

This report is encouraging because of the rather poor results which are obtained by other methods of therapy for bladder carcinoma.—*George W. Chamberlin, M.D.*

ROLINSON, E., and ROTBLAT, J. An electrical analogue of the metabolism of iodine in the human body. *Brit. J. Radiol.*, April, 1955, *28*, 191–198. (From: Department of Physics, Medical College of St. Bartholomew's Hospital, London, E. C. 1, England.)

The authors have constructed an electrical device which can be made to correspond to various phases of the iodine cycle. They have found it useful in the analysis of the results of I^{131} tracer investigations of thyroid disorders and in demonstrating the integrated functions of the different portions of the iodine cycle.

A detailed description of the electrical analogue is given in the appendix as well as a demonstration of its application, in studying a euthyroid and hyperthyroid patient.—*John P. Storaasli, M.D.*

NICHOLSON, J. P., WILSON, C. W., NEWTON, K. A., and MACLAREN, R. G. C. The use of radioiodine in the study of thyroid disorders. *Radiology*, April, 1955, *64*, 567–571. (From: Physics Department and Radiotherapy Department, Westminster Hospital, London, England.)

The authors describe procedures which have been carried out at Westminster Hospital in the study of a variety of thyroid disorders. They did not attempt accurate percentage uptake of radioiodine, but concentrated on determining its spatial distribution in areas of interest.

The first step in this investigation was the making of a plaster cast of each patient's neck. Several anatomic landmarks were painted on this cast and the surface was ruled into 2 cm. squares. Radioiodine was then given, and, after an interval of 3 hours, the cast was again fitted to the neck of the patient. Count rate readings were then taken at each 2 cm. square. From graphs of the observed count rates, isodose curves were drawn on the cast. In this way the distribution of the radioiodine lying beneath the cast was determined.

This method is presented as a simple, rapid means of determining the iodine uptake in a particular region without expensive or complicated equipment. —*A. W. Sommer, M.D.*

BULKLEY, GEORGE J., COOPER, JOHN A. D., and O'CONOR, V. J. Intraprostatic injection of radioactive yttrium chloride in the dog. *Surg., Gynec. & Obst.*, April, 1955, *100*, 405–408. (From: Departments of Biochemistry and Urology, Northwestern University Medical School, Chicago, Ill.)

Yttrium chloride containing Y with a specific activity of 1 millicurie per milligram was used. Briefly, the radioactive yttrium chloride solution was distributed through the lobes of the prostate in 19 mongrel dogs by injection from a shielded syringe. In 9 animals no attempt was made at mechanical distribution through the gland other than the use of

pressure. In the remaining 10 animals, the solution was distributed as uniformly as possible through the parenchyma by moving the needle around through the primary injection site. Urine and blood samples were obtained at twenty-four hour intervals for five days. Animals were sacrificed after one week to obtain tissues for radioactive assay, and at intervals up to three months for histologic study. The two methods of injection produced no difference in urinary excretion. The major excretion occurred in the first forty-eight hours with only small amounts eliminated during the subsequent forty-eight hours.

The level of radioactivity in the blood remained fairly constant over a 120 hour period.

After removal of the bladder, prostate, urethra, and identifiable lymph nodes, a survey with a portable Geiger-Müller counter disclosed that a large amount of radioactivity was distributed through the pelvic tissues.

Animals sacrificed at intervals varying from one week to three months showed the gross changes only in the prostate, with its immediately surrounding structures and the lymph nodes draining the prostatic area. In over half the animals marked capsular reaction with periprostatic adhesions was noted by the end of three months. Two animals were observed to have gross radiation reaction in lymph nodes as high as renal pedicle along the periaortic chain, and one was found to have gross changes in the mesenteric lymph nodes. Gross changes due to radioactive yttrium were not found in the lungs, liver, spleen, bone marrow, muscle, or kidneys at autopsy.

Microscopically, yttrium chloride caused an acute radiation reaction characterized by hemorrhage and necrosis. The acute reaction was limited to small areas in most instances. Some specimens demonstrated spread beneath the capsule rather than throughout the parenchyma, resulting in peripheral necrosis and hemorrhage. The healing process consisted chiefly of replacement of necrotic areas with fibrous connective tissue; reduction in size of the prostate began at four to six weeks and was essentially complete at the end of three months.

Some prostatic glands showed cystic dilatation and areas of pigmentation.

The urinary excretion of radioactive yttrium following intraprostatic injection did not differ significantly from that observed with radioactive colloidal gold and chromic phosphate. Apparently a considerable amount of the radioyttrium enters the urethra and bladder at the time of injection or shortly thereafter, with continuing excretion during the second twenty-four hours.

There was no damage to the urethra from exposure to this level of radioactivity as judged by histologic studies. Some yttrium was distributed to distant organs by the vascular route. The liver was the only organ which contained more than 1 per cent of the activity and the content was half that found in studies with radiogold or radioactive chromic phosphate. The remainder of the radioyttrium apparently left the prostate along lymphatic channels to the regional lymph nodes or by direct extension along the fascial planes of the pelvis. A striking feature observed at autopsy was the large number of lymph nodes showing gross radiation change. Inability to achieve uniform distribution through the parenchyma has prevented complete destruction of the prostatic gland. This same limitation applies in the use of radiogold or colloidal chromic phosphate. The greater retention within the prostate itself and the wider distribution in the regional lymphatics suggests that radioyttrium offers advantages over radiogold and radioactive chromic phosphate in the treatment of carcinoma of the prostate.—*Stephen N. Tager, M.D.*

MISCELLANEOUS

Bromley, J. F. Reflections on radiation hazards in clinical practice. President's address. *Proc. Roy. Soc. Med.*, Jan., 1955, *48*, 45–54.

This discussion is an attempt to focus attention on the responsibilities of the radiologist in advising other members of the staff as to proper protective measures when using any of the various ionizing radiations, and the responsibility of the radiologist in enlightening the general public regarding its fears generated by the publicity given to the whole subject of artificial radioactivity. The author reviews some of the early reports on radiation injuries, including two reported in 1896, only a few months after Röntgen's announcement of his discovery.

Analysis of the findings in blood counts was made, both in persons working with ionizing radiation and in a group of controls. A considerable variation in the controls was noted from day to day, both in the total white counts and in the counts of the various cell types. Some individuals were shown to have rhythmical variations in white counts over a period of years. It has been shown that arbitrary levels of safety in white counts may be misleading. An experiment was conducted to compare the sickness absence in two groups. One group consisted of workers at the atomic energy plant at Harwell, England, who had a leukocyte range of 3,700 to 7,700 per c. mm. The control group had counts in the accepted normal range of 4,500 to 13,300. The "leucopenic" group showed not only a less incidence of disease, but if they did become ill, showed a tendency for the clinical course of their illness to be shorter than did the control ("normal") group. This emphasizes the need for a clinical assessment of the general health in conjunction with the blood count.

In a discussion of the genetic effects produced by ionizing radiation, a warning is sounded against failure to differentiate between effects of radiations as a cause of gene mutations, and on the other hand of structural changes in chromosomes. A gene mutation may not become apparent for many generations, and if recessive, will not appear even though herita-

ble, unless a gene with which it is paired has undergone a similar mutation. Changes in the structure of chromosomes, however, which cause mechanical difficulties at division while permitting survival of the cell (instead of causing death as more frequently happens) will produce heritable changes (in the offspring) analogous to gene mutations but often of a dominant character. Such "chromosome abnormalities" may cause damage or infertility in the first generation offspring, and in this respect are far more detrimental than induced gene mutations, which are usually recessive in character.

Other experiments and observations are reported including the radiation hazard for nurses in radium wards, and the increased hazard involved in bunching patients undergoing radium therapy for gynecological conditions. A number of graphs and charts are included.—*D. S. Kellogg, M.D.*

Stehney, A. F. Radium and thorium x in some potable waters. *Acta radiol.*, Jan., 1955, *43*, 43–51. (From: Argonne National Laboratory, Lemont, Ill.)

This study was made in order to obtain data on natural levels of radioactivity and to help locate potable water supplies having an unusually high radium content as part of a current research program on the toxicity of radium. It was found that the concentration of radium in surface water or shallow wells in the Chicago area is low,—less than 0.1 $\mu\mu$c. per liter. Water from wells which penetrate into the Galesville formation of sandstone of Cambrian age, at a depth of approximately 1,500 feet, near Chicago, Illinois, was found to contain from 3 to 8 $\mu\mu$c. Ra-226 per liter and about the same amount of thorium x. This is believed to be the highest authenticated concentration of radium found to date for any drinking water in the United States. The combined population of the communities using these wells is over 100,000. Drinking water from Frankfort am Main, Germany, was found to contain less than 0.3 $\mu\mu$c. Ra-226 per liter.

Much higher values for the radium content of some spring and mine waters have been reported by others. However, it is recognized that some of the earlier values reported as radium were in reality for radon, the activity of which is often several orders of magnitude greater than that of radium in the same water sample. An extreme case of the disparity of radium and radon concentrations in a single water source was reported by Kuroda and Yokoyama, who found a Masutomi Spring (designated as A49) in Japan to contain 1.33×10^6 $\mu\mu$c. Rn per liter and only 8 $\mu\mu$c. Ra per liter. The highest radium content reported by Kuroda and Yokoyama was 76 $\mu\mu$c. per liter for Masutomi Spring A5.

The radium content of public water supplies is usually much lower. The highest concentration found by Hursch recently, in a large number of samples of drinking water from the United States, was less than 2 $\mu\mu$c. Ra per liter. A discussion of analytical procedures and detailed charts of water analyses are included.—*D. S. Kellogg, M.D.*

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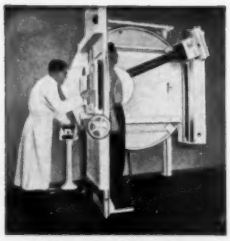

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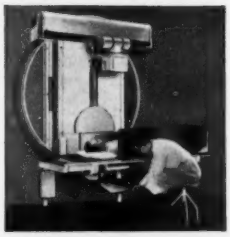

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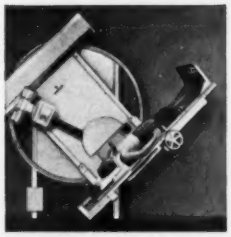

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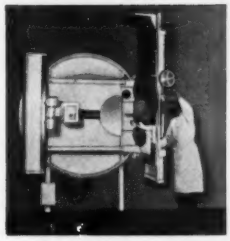

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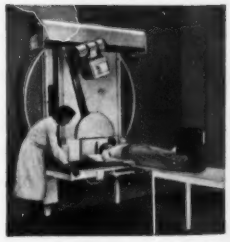

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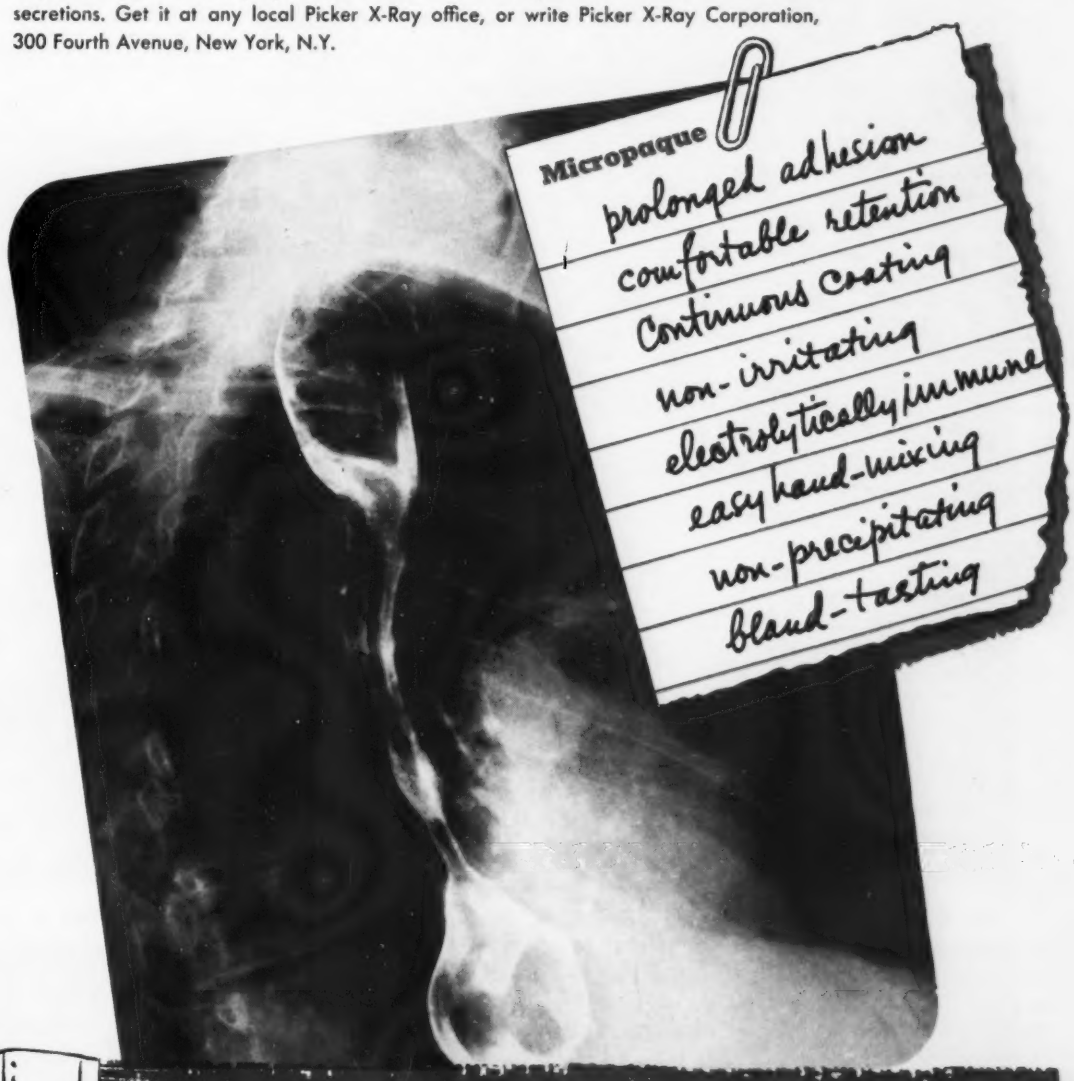

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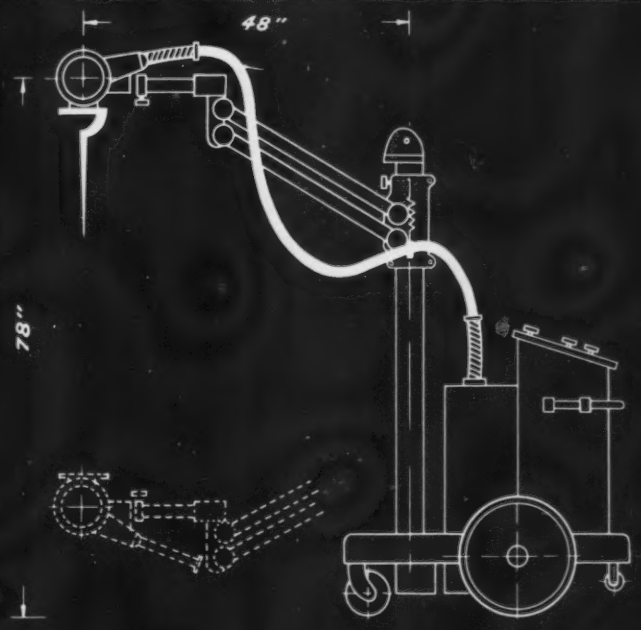

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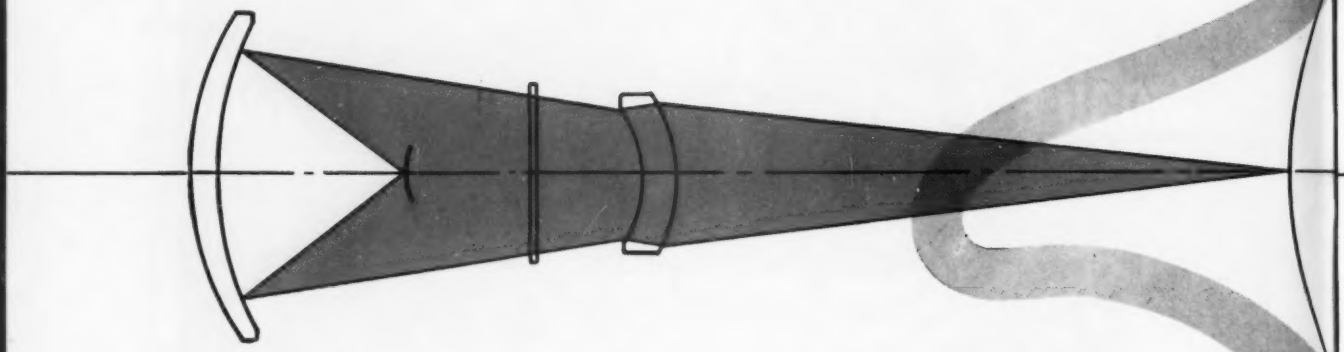

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Miokon Sodium

Brand of Sodium Diprotrizoate

miokon® SODIUM

A CONTRAST MEDIUM

ESPECIALLY FOR

INTRAVENOUS UROGRAPHY

BY Mallinckrodt®

THE MAKERS

OF UROKON®

SODIUM

*Trademark

CLINICAL REPORTS

ON MIOKON SODIUM 50%
FOR INTRAVENOUS UROGRAPHY

MIOKON Sodium has been studied by a total of fifteen clinical investigators in 2088 cases of intravenous urography.
The pilot clinical study was carried out at the Washington University School of Medicine under the supervision of Dr. Charles H. Nicolai of the Department of Urology.[1] In this pilot study of 1138 consecutive cases examined with MIOKON Sodium, this group observed a total of only 10% reactions and obtained 96.5% diagnostic pyelograms. Later Dr. Nicolai reported:

"Particularly notable in our series of 1500 cases with MIOKON was the low incidence of minor side reactions such as nausea and arm pain. In addition, diagnostic pyelograms were obtained in over 96% of the cases, a percentage higher than previously recorded with any contrast medium in this hospital. Although our studies indicate that MIOKON is one of the most satisfactory media available, it should be used with care since all contrast media produce an occasional vasomotor reaction."[1]

In further clinical studies, MIOKON Sodium was evaluated by fourteen additional clinical groups. These investigations included 950 cases. Diagnostic pyelograms were obtained in 95.4% of these cases; 14.2% reactions were observed.[2]
Most observers reported that the minor side effects which occurred following injection of MIOKON Sodium were mild and fleeting and caused no particular distress.

1. a) Nicolai, C. H.: MIOKON, A Preliminary Clinical Report on a New Intravenous Urographic Medium, Missouri Medicine, *52*:703. (September, 1955).
   b) Nicolai, C. H.: (Private Report dated July 19, 1955 on 1138 cases).
   c) Nicolai, C. H.: (Private Communication dated August 31, 1955 on 1500 cases).
2. Data in the files of the Medicinal Chemical Development Department, Mallinckrodt Chemical Works.

Intravenous urogram made with MIOKON Sodium 50%.
Shows hydronephrosis in a horse-shoe kidney.

miokon

...developed as a result of the overall excellence of **UROKON SODIUM**

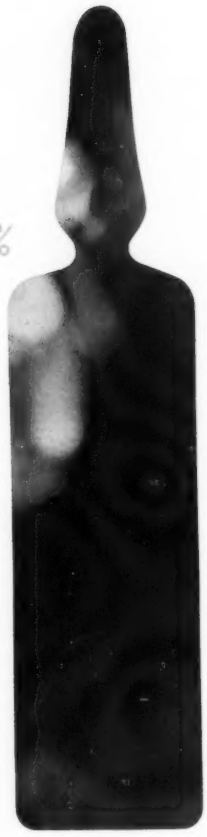
miokon SODIUM 50%

For some techniques, most contrast media available to urologists and radiologists prior to 1950 were not up to today's standards. Too often, diagnostic films were obtained in a relatively small percentage of cases; reaction rate was high. With the introduction of Urokon 30% in 1950, it became possible to obtain diagnostic pyelograms in a high percentage of cases (89% reported by investigators) and reactions dropped to only 19% of cases observed.

The discovery of Urokon*, with an entirely different basic structure, changed the direction of development work on contrast media. The new structure-activity relationship which was revealed in the overall excellence of Urokon led to the development of Miokon.

*Sodium salt of
3-acetylamino-2,4,6-triiodobenzoic Acid.

miokon SODIUM 50%



PROVIDES **96%***

DIAGNOSTIC FILMS

WITH MINIMUM SIDE REACTIONS

*A series of 1500 cases actually yielded 96% diagnostic films. Any further improvement of the percentage of diagnostic films is difficult to foresee, since most contrast media available today fail only in patients having poor renal function or because of other factors independent of the media.

- FEW SIDE REACTIONS
- NOTABLY SAFE
- LOW VASOMOTOR RESPONSE
- HIGH OPACITY TO X-RAYS

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For some techniques, most contrast media available to urologists and radiologists prior to 1950 were not up to today's standards. Too often, diagnostic films were obtained in a relatively small percentage of cases; reaction rate was high. With the introduction of Urokon 30% in 1950, it became possible to obtain diagnostic pyelograms in a high percentage of cases (89% reported by investigators) and reactions dropped to only 19% of cases observed.

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*Sodium salt of
3-acetylamino-2,4,6-triiodobenzoic Acid.

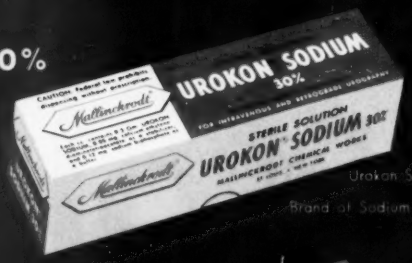
UROKON®

still the most Versatile

CONTRAST MEDIA OF ALL

UROKON® SODIUM Sterile Solution **30%**

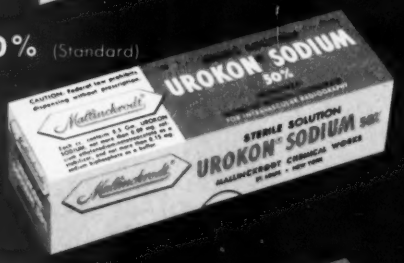
widely accepted for
INTRAVENOUS UROGRAPHY (in routine cases),
RETROGRADE PYELOGRAPHY and
CHOLANGIOGRAPHY



Urokon Sodium
Brand of Sodium Acetrizoate

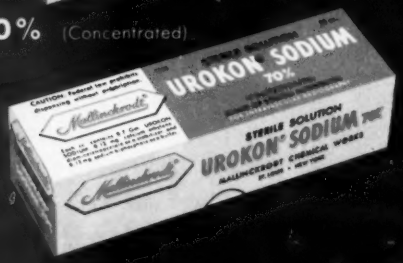
UROKON® SODIUM Sterile Solution **50%** (Standard)

for
INTRAVENOUS UROGRAPHY (in routine cases).
May also be used for CHOLANGIOGRAPHY
and (in dilution) for RETROGRADE PYELOGRAPHY



UROKON® SODIUM Sterile Solution **70%** (Concentrated)

for
INTRAVENOUS UROGRAPHY (in difficult cases),
ANGIOCARDIOGRAPHY, CHOLANGIOGRAPHY,
TRANSLUMBER ARTERIOGRAPHY, NEPHROGRAPHY
and (in dilution) for RETROGRADE PYELOGRAPHY



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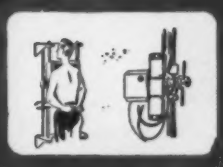
currents up to 1000 mA

Super DLX is designed to meet all the requirements of the radiologist as regards techniques.



The apparatus has been designed for all radiologists demanding not only a high powered unit but also one which provides dependable operation.

Kilovoltage is fully compensated: the mAs product is pre-indicated. For any combination of exposure factors, an automatic control system shows if the focus will be under-or fully loaded, and will prevent overloading.

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Tracerlab's new Tracer-Scanner automatically maps the uptake and location of radioactive material in certain organs, such as the thyroid, and provides a two-dimensional graph which clearly defines the distribution of the isotope. The complete unit is mounted on a table which easily rolls over a hospital bed. It carries a scaler and a highly-collimated scintillation detector which traverses the body area being scanned.

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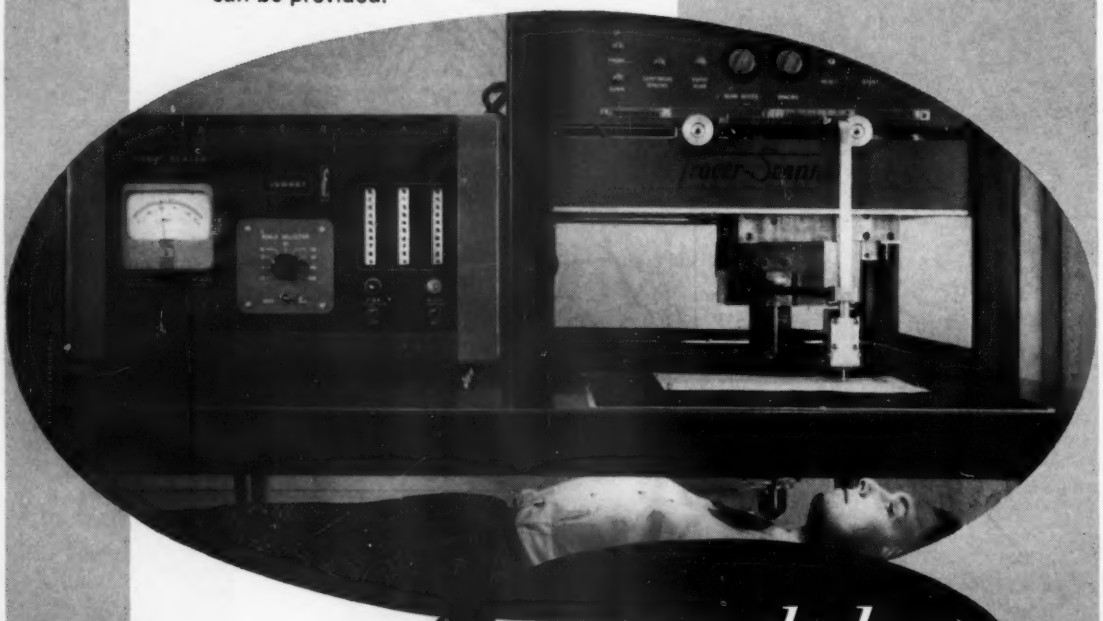

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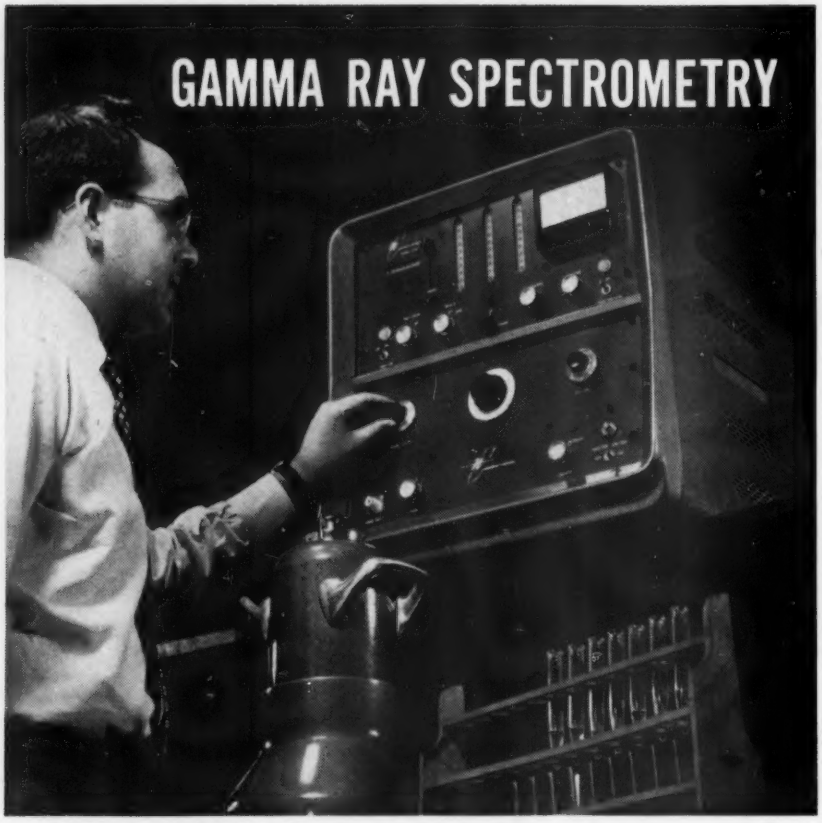
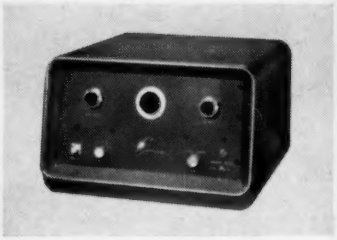
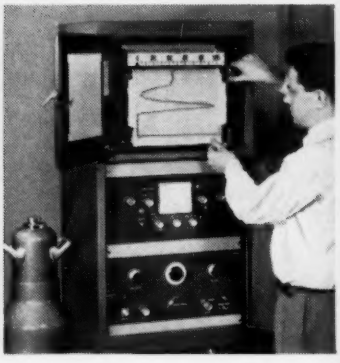


Gamma emitting samples inserted into a scintillation well counter are counted by Radiation Analyzer and standard scaler. Adjustable "window" of Analyzer is set to count only pulses that fall within a pre-selected portion of the energy spectrum, thus virtually eliminating "background".



Analyzer is available separately as an economical accessory for laboratories already having a scintillation detector and a scaler or rate-meter.



Automatic scanning and recording of spectrum energies is accomplished by Analyzer, rate-meter, recorder and scintillation counter. Gamma energy spectrum of Cesium-137 is shown on recorder chart.

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The advantages of pulse-height selection for background and scatter-error reduction, multiple tagging measurement and energy analysis are now available for every radioisotope laboratory. The Radiation Analyzer, connected directly to your present scaler and scintillation counter, incorporates in a compact single-chassis design a non-overloading linear amplifier, pulse height discriminator and extremely stable high voltage regulator. Circuitry has been designed for simplicity, stability and high reliability.

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(WHITEHOUSE)[1]

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"Even more gratifying has been the visualization of the bile ducts which occurred in about two-thirds of the cases in which the gallbladder was visualized."

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(TWISS, et al.)[4]

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SUPERIOR ORAL CHOLECYSTOGRAPHIC MEDIUM

Average adult dose:

6 Telepaque tablets orally. For persons of thin or medium build under 150 lb., 4 tablets are usually sufficient. Tablets should be taken with at least one full glass of water.

SUPPLIED:

Tablets of 0.5 Gm., individual envelopes of 6 tablets, boxes of 5 and 25 envelopes. Bottles of 500 tablets.

1. Whitehouse, W. M. (University Hospital, University of Michigan, Ann Arbor): *Surg., Gynec. & Obst.*, 100:211, Feb., 1955.
2. Shehadi, W. H. (New York Polyclinic Medical School and Hospital): *Am. Jour. Roentgenol.*, 72:436, Sept., 1954.
3. Carroll, W. H.; and Riley, G. M. (Shreveport): *Jour. Louisiana Med. Soc.*, 106:141, Apr., 1954.
4. Twiss, J. R.; Gillette, Lee; Beranbaum, S. L.; Poppel, M. H. (New York); and Hanssen, E. C. (Pasadena): A.M.A. *Arch. Int. Med.*, 95:59, Jan., 1955.

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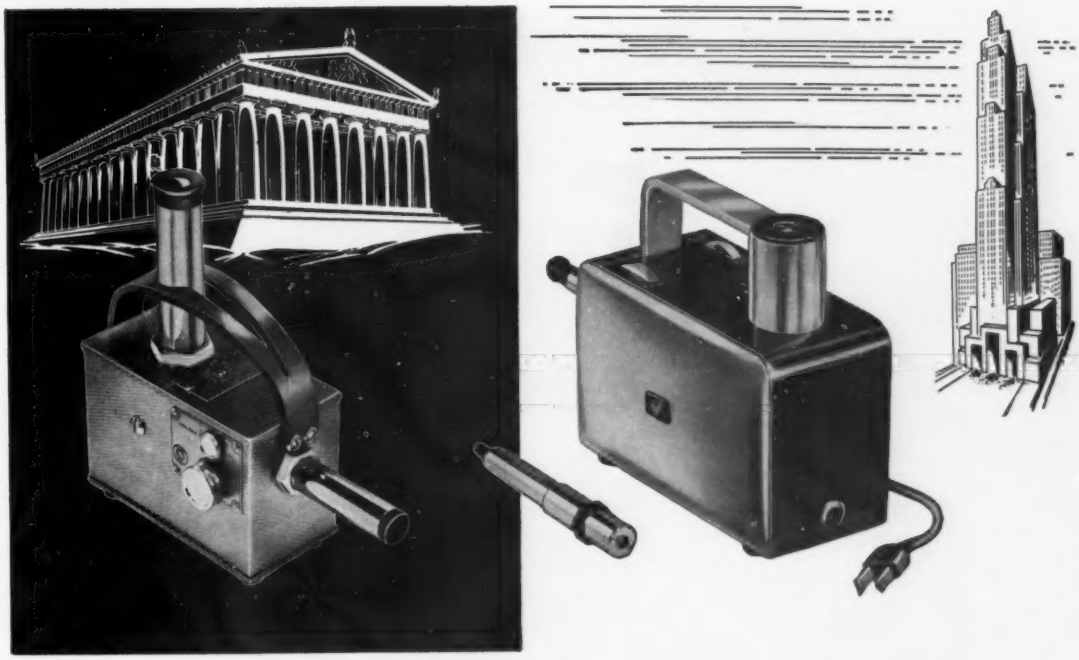

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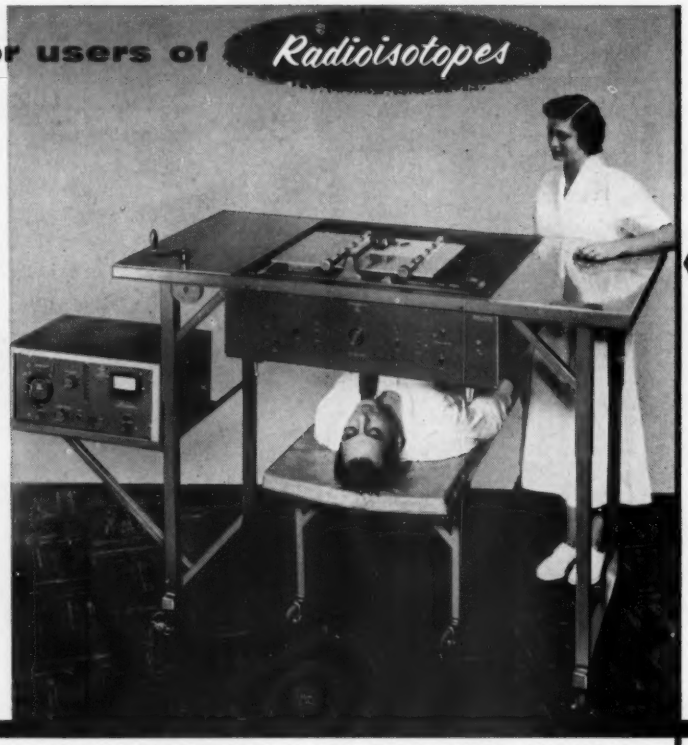

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Children's Radiographic Technic

STUDY NO. 10

NASOPHARYNX: LATERAL VIEW

This view is useful for demonstration of excess lymphoid tissue (Adenoid). It is taken with the patient supine, tube horizontal and cassette perpendicular. Expose at passive expiration with cooperative patients and at inspiration with uncooperative little children. Mouth must be closed or held firmly with webbing strap beneath patient's chin to prevent mouth breathing.

TECHNIC Central Ray is directed through the zygoma to the cassette which is placed in a perpendicular position.

Age Group	K.V.	M.A.	M.A.S.	Dist.	Time	Alternate Time 100 M.A.	Remarks
up to 3 mos.	58-61	200	3.33	40"	1/60	1/30	med. cone 4-6 inch
up to 2 yrs.	58-61	200	5.	40"	1/40	1/20	med. cone 4-6 inch
2 to 5 yrs.	61-64	200	5.	40"	1/40	1/20	med. cone 4-6 inch
5 to 7 yrs.	58-61	200	6.67	40"	1/30	1/15	med. cone 4-6 inch

NOTE: Technical factors were obtained using par-speed screens.

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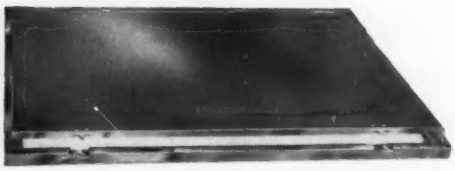

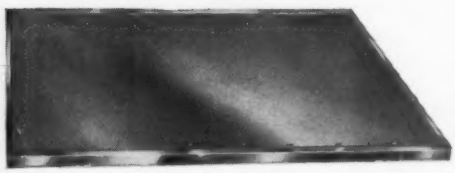

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[*Marks, J.H.: New England J. Med. 250:999 (June 10) 1954.*]

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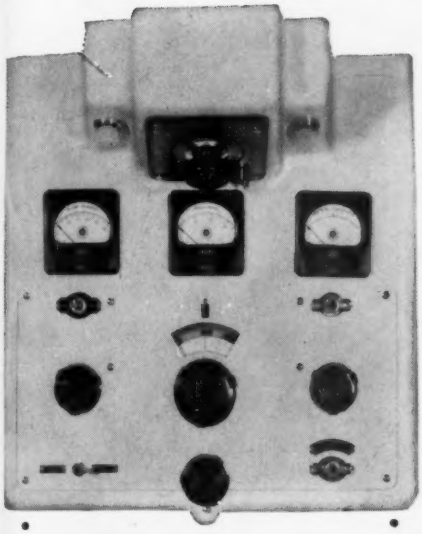

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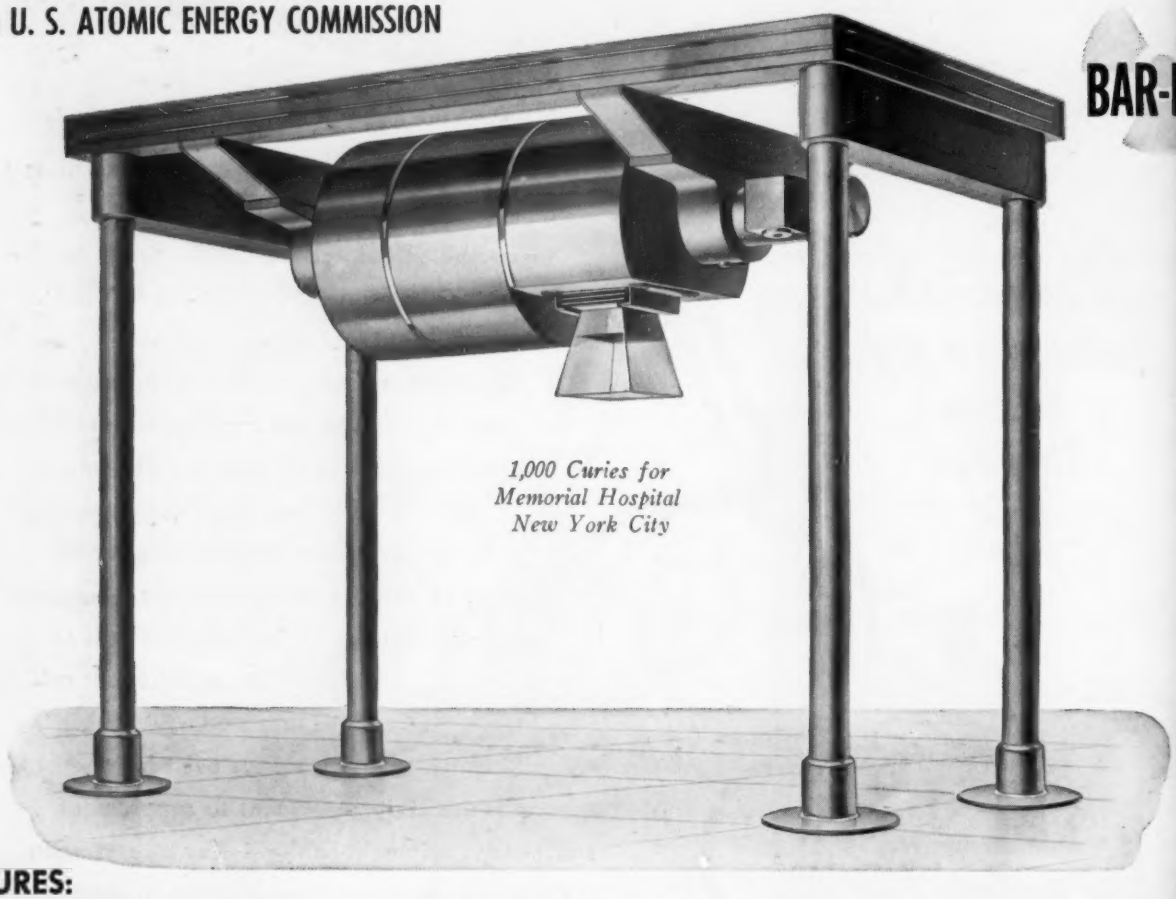

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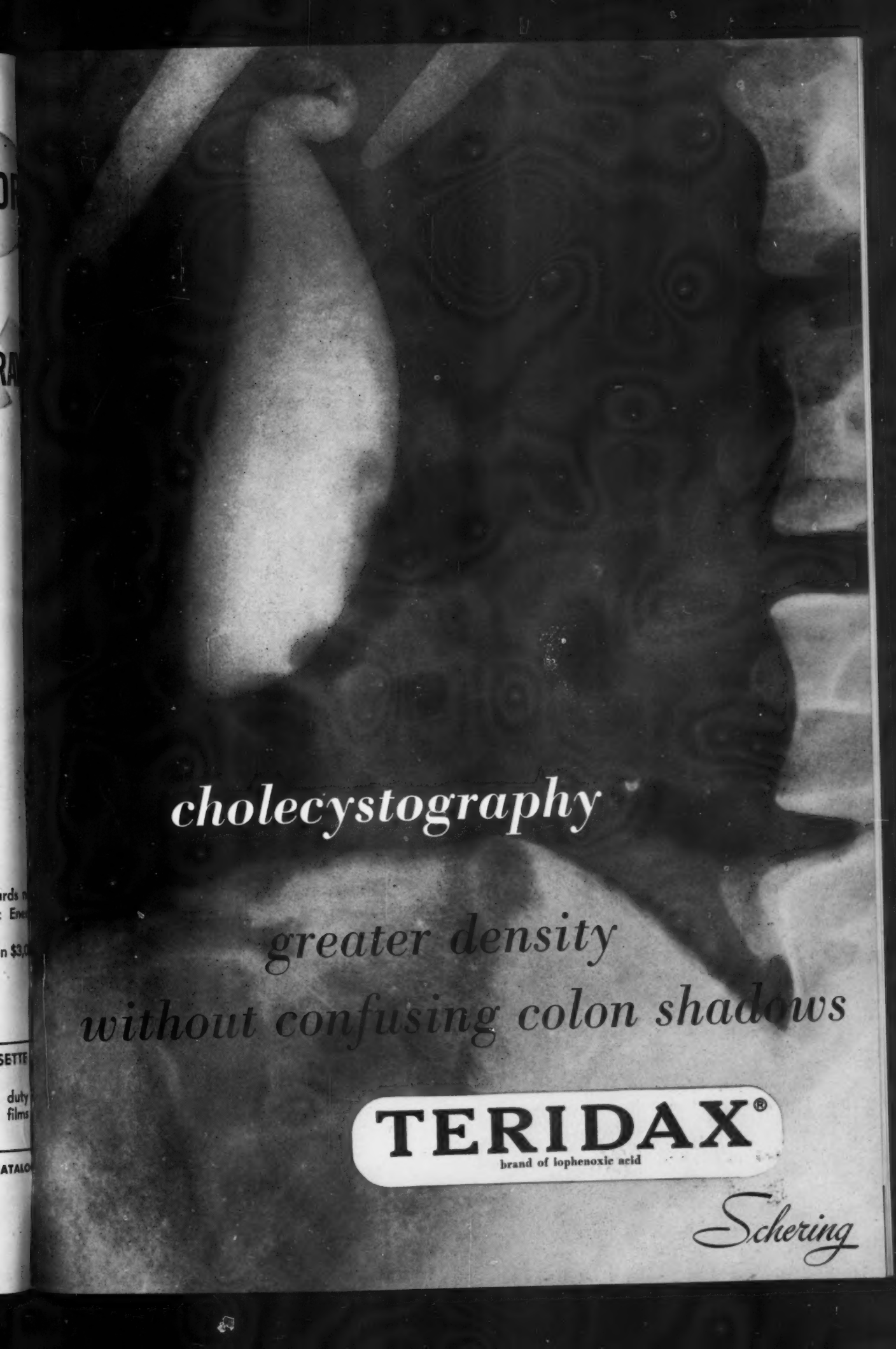
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RADIOGRAPHS: A-P preoperative and postoperative.

COLOR PHOTOGRAPHS: Surgical repair—6 views; this page and page following.

1. Left leg showing adherent scar over tibia. Large pedicle flap graft from opposite leg extends over to fibula.

2. Exposed *peroneus longus* muscle and fibula. Scarred extensor muscles being retracted from tibia and fibula.

3. Fibula sectioned about 4 centimeters above *malleolus*, and is being mobilized for insertion into tibia.

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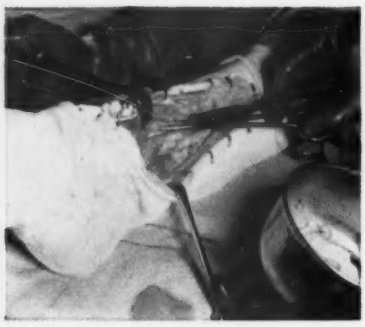
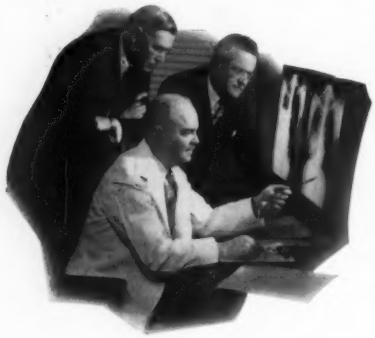


COLOR PHOTOGRAPHS: Continued from preceding page.

4. Foot displaced laterally correcting varus. Fibula being fastened into lateral side of tibia.

5. Stainless steel screw holds fibula firmly in cavity. Bone fragments packed into to insure solid healing.

6. Space between fibula and tibia above nonunion packed with autogenous cancellus bone and cortical bank bone.



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Troisième Partie. Synthèse bio-clinique

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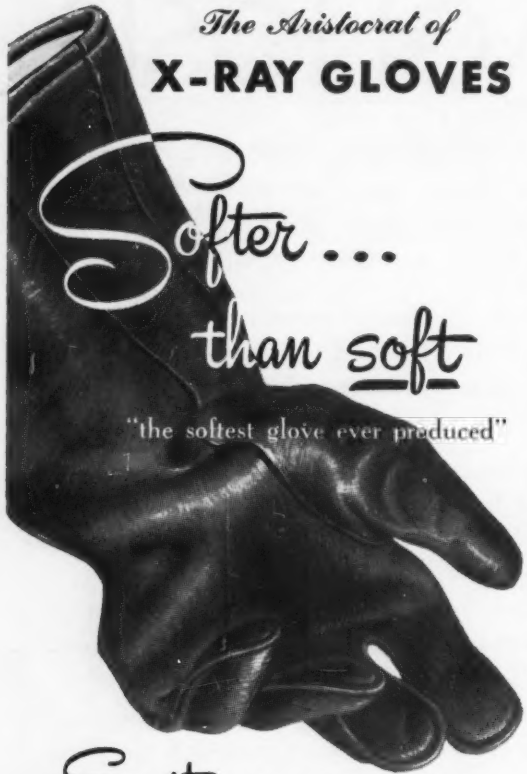

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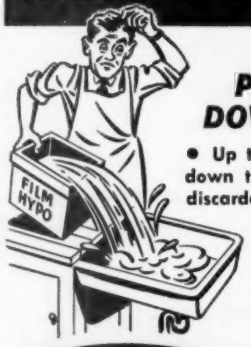

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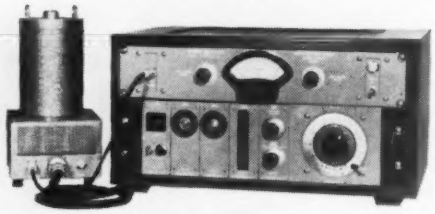

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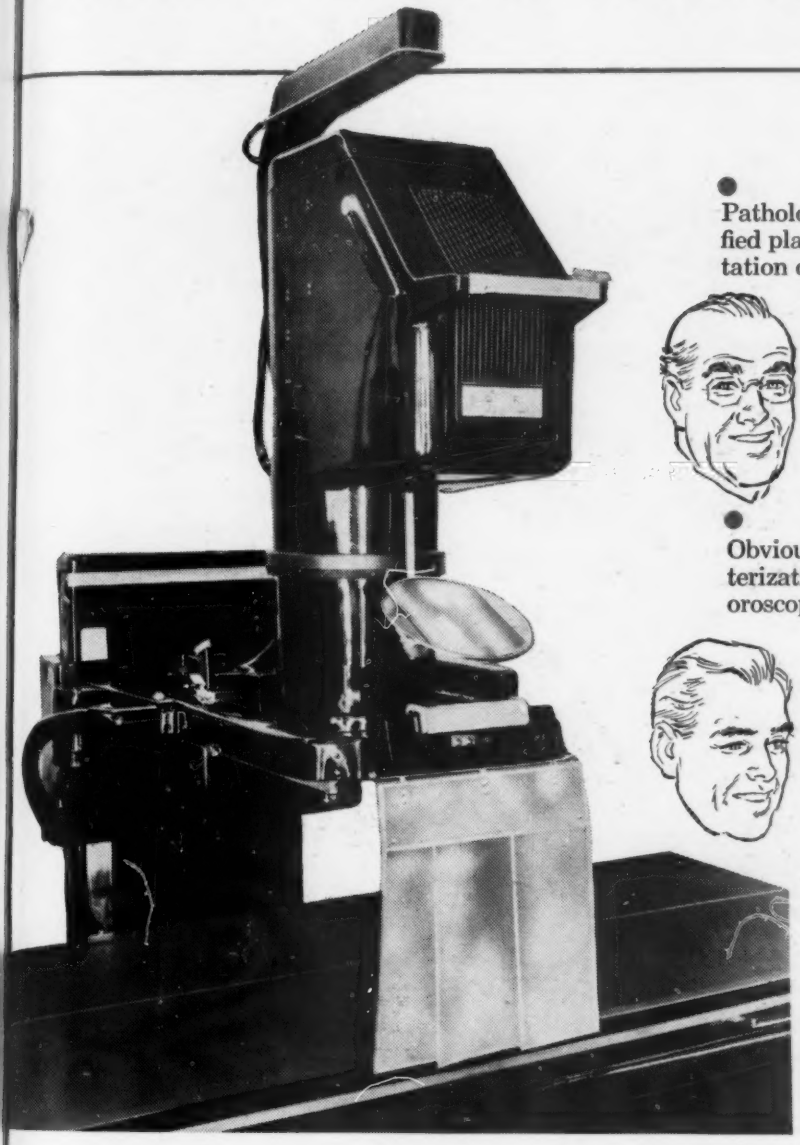

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